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The Sir Richard Stawell Oration.[1]

CHURCHILL AND HIS CONTEMPORARIES.

By THE RIGHT HONOURABLE R. G. MENZIES, C.H., Q.C., M.P.,

Prime Minister of Australia.

IN common with all of you, I remember the name and life and work of the late Sir Richard Stawell with deep respect. He was a man of high character, of clear mind, and with a deep sense of justice.

Great physicians are not always well known to the public, nor is the quality of their work always properly appreciated. It is one of the splendours of the medical profession that its greatest men have not sought public notoriety, but have without restriction devoted their talents to the service of mankind, privately, quietly, their greatest technical achievements known only to their peers, and their greatest human achievements known only to their patients and their patients' families.

When I was offered the honour of delivering the Stawell Oration I at first demurred on the intelligible grounds that there were so many more men who knew so much more about him; and that, in any event, I was not medical, and could therefore not speak of his work with an informed and discriminating judgement. I was persuasively reassured by being told that the medical profession liked every now and then to listen to a speech by a layman, and that I could quite readily and acceptably speak upon a topic unrelated to medicine; except that, being in commemoration of Richard Stawell, it might properly refer to men of courage, character, ability and consequence. Whether this concession to the laity was the product of that sadistic spirit which must occasionally invade the mind of even the most humane of medical men or was due to a desire to get away from "talking shop" I am not to judge. But I have assumed the best in my own favour and have therefore undertaken to speak to you about "Churchill and his Contemporaries"; all of them men who would have been delighted with Richard Stawell and would have found so many matters in common with him in the realm of the mind and of the spirit.

As you may know, I have for many years now been engaged in public life. Sometimes the people have been good enough to approve of that fact, and sometimes their rapture has been modified. But by and large they have been generous to me, so that in the result I have been able, over a period of twenty years, to represent this country abroad on many occasions and to achieve the

[1] Delivered at a meeting of the Victorian Branch of the British Medical Association on October 8, 1955.

acquaintance and, in some cases, the close personal friendship of some of the great men of this era. There is a strange quirk in human nature which I commend, if that be necessary, to the consideration of the psychologists and psychiatrists among you. It is this. When we are very young and we read our history, we visualize the great men of the past as giants. Their very shadows appear to be enormous as they pass across the dim and distant landscapes of history. I have lived long enough and had sufficient experience to find that historic giants are quite human, that for the most part they are quite intelligible, that in many ways they think and behave just as we do, and that one must discern their greatness, not by standing with dumb amazement before them, but by trying to discover what special quality each of them has which marks him out for fame.

In the result I have found both the great Churchill and his great contemporaries refreshingly human and indeed intelligible to people like myself for the bulk of their time.

The idea of an incomprehensible genius, which once obsessed my mind in contemplating the noble figures of the past, has long since deserted me, except in the presence of eminent mathematicians, nuclear scientists, and second-year medical students. Genius in the current affairs of men usually expresses itself in the most comprehensible terms. The whole of my experience has indeed confirmed me in my very early belief that lucidity is one of the cardinal virtues, and that people who understand their business can usually explain it reasonably clearly to normally educated and intelligent men. But I would not have you believe that this means that for me the romantic conceptions of youth have given place to a dry cynicism. About so many of the great I still remain in the frame of mind of Browning when he wrote that simple but moving verse:

> Ah, did you once see Shelley plain,
> And did he stop and speak to you,
> And did you speak to him again?
> How strange it seems, and new!

This preliminary excursus is designed to persuade you that, if in the course of my later remarks I speak in somewhat positive terms about some of the famous men of our era, you are not to assume that I do not bow before them or profoundly admire their contribution to the welfare of man. Neither praise nor criticism from somebody like myself will, I trust, be regarded as impertinent; the truth is that, unless those of us who live on the plains but have occasionally visited (strictly as guests) the slopes of Olympus are prepared to set down some human remarks about great men, contemporary history when it comes to be written will be falsified by the propagandists and by those frequent biographers whose picturesqueness and dogmatism are in inverse ratio to their knowledge. And so, with your permission, I begin with the great Churchill himself.

You already know a great deal about him. He has been a soldier, a turbulent and frequently unsuccessful politician, a leader of "lost causes and impossible loyalties", rejected at somewhat more than my own age, and ultimately the idol of the world. And all the time he has written; his books have been read across the world. His command of what I will call nervous English is unequalled in our generation. He has explained himself as few men have done. And yet his human qualities, without which his soaring imagination and command and eloquence could not have availed so much, are for most of us a deep mystery.

If I were to say to you that as I have seen him, he has had the wisdom of venerable and embattled statesmanship, in action like an army terrible with banners, and, off duty, and sometimes on duty, the chuckling spirit of a school boy; a remarkable capacity for political hostility and a much more remarkable capacity for the most endearing personal friendship and goodwill; you would begin to see that the roots of his genius are deep in a soil which produces humour and understanding and good temper and bad temper and all those oddities which go to make up the English character and occasionally, as in his case, produce the most superb genius.

I have written and said so much about him in recent years that I must avoid repeating myself. I have known him from time to time for many years. I sat with him longest in the War Cabinet in the first part of 1941, when the German raiders came over every night. And since the war, in quieter but difficult days, I am honoured to say that I have enjoyed in large measure his personal association and friendship and goodwill.

His political opponents have frequently felt the lash of his tongue. But it has never been a crude lash. Indeed, I have sometimes felt that his victim in the House of Commons felt that it was a singular honour to be attacked by him. That is one of those inexplicable things that perhaps only a politician can understand.

Could I give you one illustration of the way in which his mind and body responded to the challenge of the war?

I am thinking of one week-end night at Chequers in about March of 1941, when General de Gaulle was in England, and when Churchill, de Gaulle and I sat together at dinner in this famous old house in Buckinghamshire. De Gaulle was by common consent a brilliant soldier; but it is not easy for a brilliant soldier to become quite suddenly skilled in the politics of a French Resistance, in the economics that go with politics, or in the tactful handling which, believe it or not, is one of the essentials of international relations. In brief, de Gaulle was as long as the average Frenchman is short; in place of the celebrated French *esprit*, he possessed a somewhat sombre appearance and smiled with difficulty. At the time of which I am speaking, his English was, to say the least of it, "sketchy"; on the whole, conceivably inferior to my French. The conversations occurred in French. Winston's French is magnificent, but it is not French. *"C'est magnifique, mais ce n'est pas le français."*

I gather from my friends in London that the celebrated Birkenhead had once said of Winston's French: "You know, I greatly admire Winston's French. It is the only French I have ever been able to understand and the odd thing is that the French appear to be able to understand it also!"

In this setting I was, I confess, being a little wickedly provocative. Every time harmony appeared to be breaking out I would throw in some vulgar observation about Dakar—a subject upon which Winston and I had exchanged cables and on which de Gaulle had somewhat turbulent views. It was a remarkable experience. We adjourned into another room. Churchill and de Gaulle walked up and down, delivering homilies at each other. I sat back with the comfortable feeling that I was witnessing a fascinating phase of history. By 2 o'clock in the morning de Gaulle very sensibly decided to go to bed. I decided, for no reason that I can sensibly recall, to stay up.

The great man himself went to bed at 3 o'clock in the morning; but before he did so he went into the little corner study at Chequers and rang up Bomber Command and Fighter Command to get the reports of the day. What he had to say to them on their reports was all compact of encouragement, rebuke, fire, criticism, what you will. Next morning I was hugging my pillow at some rational hour, and arriving for breakfast reluctantly at 9 o'clock, only to find that at 7 the Prime Minister had received his dispatches, had sat up in bed with some black coffee and a large cigar, and was busy dictating the directives of the day.

We do not see men like this in every generation, nor indeed does the world see too many in a century. I must confess that over the years I have never known Winston to observe any of the rules of health. Yet his amazing mental fire must have been associated with a remarkable physical tenacity. The two things worked together, partly because they were born in him and partly because, consciously or unconsciously, he cultivated them, using adversity to strengthen them.

The trite saying that "the English lose the battles, but win the wars"; Philip Guedalla's epigrammatic explanation of the great Duke of Wellington's subsequent loss of nineteenth century reputation, that the English prefer

their heroes to be slightly unsuccessful, to retreat gloriously to Corunna or die in the hour of victory at Trafalgar; these are not irrelevant. It is indeed part of the legend of our race to come from behind and to snatch victory from defeat. In my war-time association with Winston Churchill, I caught, paradoxically, a few echoes of this legend. Not that the great man was ever defeatist. Far from it. Never was there a leader more unwilling to contemplate a defeat or acknowledge a reverse. But I have seen him and heard him discuss a current situation, building up the intensity of the problem, tearing away wishful thinking, only to proceed from there literally to fight his way through the problem to a point at which all of us who were his hearers not only believed but knew beyond peradventure that, given courage and energy and endurance, victory was ours.

I could talk to you for a long time about him, about his charming and magnificent wife, and about his family. But I must resist this temptation, because I must turn for a little to some of his contemporaries in order to disclose to you my deep-seated belief that great individual powers are not a freak of nature, but form part of a pattern of greatness in any country or generation. After all, even in the spacious days of great Elizabeth, Shakespeare was not a lonely figure in the superb renaissance of poetry and drama. If he had not lived at all, we would be reading the other Elizabethan dramatists much more than we do. Trees grow tallest in a tall forest, and so, believe me, Churchill has had great contemporaries. He has himself in a notable book written of some of them. If you go back home and reread "Great Contemporaries" you will find not the heartless cut and thrust of political controversy, but great men written of justly, generously and affectionately.

Birkenhead's place in history is no doubt a matter of controversy. Quite plainly his talents were greater than his achievements, and yet Churchill wrote of him the most splendid epitaph that mortal man could wish:

> Some men when they die after busy, toilsome, successful lives leave a great stock of scrip and securities, of acres or factories or the goodwill of large undertakings. F.E. banked his treasure in the hearts of his friends, and they will cherish his memory till their time is come.

But let me for a few minutes go back before Churchill.

I have known six Prime Ministers of Great Britain. Two of them, Mr. Attlee and Sir Anthony Eden, are still on the active political scene, and therefore, though I could speak of each of them with deep admiration and affection, it would be an impertinence for me even to appear to sit in judgement upon them.

But three of them preceded Churchill — Ramsay Macdonald, Stanley Baldwin, Neville Chamberlain. Each in his day enjoyed wide popularity. Churchill, thank God, still does. But three of them went out of office, if not unhonoured, at least unsung.

Now I entertain what some of my friends regard as the eccentric belief that Macdonald, Baldwin and Chamberlain were great men who rendered certain vital and abiding services to their people, and that Churchill, with all his genius to command and inspire, could not have done everything that he did but for their work.

Each was at one time, no doubt, over-praised. But each has subsequently been over-condemned. It does little credit to our good sense that we should swing about so wildly in our judgements, treating today as mere folly our wild enthusiasms of yesterday. After all, if our superficial emotions are our only guide, we have no more assurance that we are right today than we were wrong yesterday.

When I first met him, in 1935, Ramsay Macdonald was long past his best. He had become tired and old, and addicted to rather vague and meaningless phrases. A leonine and handsome appearance, a fine poise and a rich voice, with occasional flashes of international insight were all that seemed to remain of a man whose personality and force of character must have been great to lift him from a position of almost universal unpopularity and, indeed, opprobrium during the First World War to No. 10 Downing Street only a few years later.

My own contact with him was sketchy enough—a few meetings in London, a visit to Chequers; but I count his brilliant son, Malcolm Macdonald, now British High Commissioner in India, as a warm though now a geographically distant friend. I remember Malcolm saying to me one day in London (I know he will not mind me quoting him):

> You did not know my father at his best. I can recall him standing on the tail of a truck, by torchlight, speaking to a thousand miners with such power and appeal that the tears made white furrows down their faces. As a spokesman for the under-dog, as a denouncer of social and industrial injustice, he was tremendous and unforgettable.

It is easily believed. England was (and is) a traditionally conservative place. It was Ramsay Macdonald who, with fire and great political skill, brought the Labour Party from a small obscurity to the seat of government. He formed and led the first Labour Government. This (and here I state the point of my narrative) was not a mere accidental or transitory political triumph. It gave to organized Labour, for the first time, a sense of power and therefore, inevitably for sensible men, a feeling of political responsibility.

It would surprise me if the future historian, battling his way through all the partisan records, did not come to the conclusion that but for the work of Ramsay Macdonald there might have been no instant place for a socialist Ernest Bevin as Minister for Labour in a Conservative-led War Cabinet in the Second World War. The magnificent cooperation of 1940-1945 proceeded from a consciousness in the industrial unions and among politically organized wage-earners not only of the necessities of their country, which they knew clearly enough, but also of their own national powers and responsibilities. The British National Government of 1940-1945 gave a lead and direction more authoritative than could have been provided by any one-party administration.

Stanley Baldwin's political reputation is today surrounded by clouds and darkness. The current picture of him is that of an indolent and not very gifted man, sucking at his pipe or inspecting his pigs, oblivious of the state of Europe or the rising menace of Hitler, ignoring the eloquent warnings of Churchill, allowing his country to go on, unaware and unprepared, to the very edge of the abyss.

Some of the lines in this picture are, alas, true enough. Some are fantastically wrong. I saw a good deal of Baldwin in those years. He was a plain and solid Englishman, of great personal friendliness and charm, an easy and indeed magnetic talker over the breakfast table, a supreme Parliamentarian in the House of Commons.

He was a poet at heart, a master of that kind of simple and moving speech which best expresses the underlying passion of the Englishman for his own countryside, its history, its form, its familiar colours and smells.

> To me, England is the country, and the country is England. And when I ask myself what I mean by England, when I think of England when I am abroad, England comes to me through my various senses—through the ear, through the eye, and through certain imperishable scents . . . The sounds of England, the tinkle of the hammer on the anvil in the country smithy, the corncrake on a dewy morning, the sound of the scythe against the whetstone, and the sight of a plough team coming over the brow of a hill . . . And above all, most subtle, most penetrating and most moving, the smell of wood smoke coming up in an autumn evening, or the smell of the scutch fires.

At more than one period of domestic political crisis his conduct was cool, shrewd and successful. The general strike of 1926, trouble on the coalfields, the unprecedented problems of the abdication, were all handled by him with skill and a just understanding of underlying British opinion.

What was the secret of these successes, so sharply contrasting with his chronic failure to realize or deal with the menace arising in Europe?

The answer is that he was an Englishman of great character and talent, but a provincial Englishman. Europe mystified him; he was never attracted to its history or its

problems; he probably illustrated to perfection the old and true proverb about the rural Englishman, that for him "the Negroes begin at Calais". Steel-master Baldwin might be, by force of circumstances. But at heart he was of the English country; ready to recall his people to its beauties; possessed in rare degree of the faculty of invoking a sense of national unity. It was this sense of unity which defeated the general strike, which at one stage averted grave trouble in the coal mines, which plucked out of the thorns of the abdication the flower of an actually strengthened Crown.

The historian's balance may, for aught I know, weigh down against Stanley Baldwin. But the superb national unity with which Great Britain went to war against odds on September 5, 1939, owed not a little to the man who had nurtured it in the deep and simple pride of his people.

Neville Chamberlain succeeded him at a time when the average Englishman still did not accept the inevitability or even the real probability of war. Chamberlain was the son of the great champion of tariff reform and Imperial preference. His family and political background was industrial. But, as in the case of Baldwin, he was at heart a countryman. He would turn from the complexities of a budget to a week-end whipping a remote stream, identifying obscure plants during some woodland ramble, or listening with joy to the song of a bird (and a bird he knew) in a hedged lane.

I could never understand why he was so little understood. "He would have made a good Lord Mayor of Birmingham—in a bad year!", said the mordant Birkenhead. Yet, as I shall try to show, he rendered services to his country certainly no less remarkable than those of Birkenhead himself.

"A mere accountant!", said another critic. Yet on two occasions I sat in the gallery of the House of Commons and heard him deliver a budget speech with such clarity, point and dramatic sense that I shall always regard them as among the greatest budget speeches I ever heard.

And what of Munich?

We might as well admit (in British countries at any rate) that, when the Prime Minister who had never flown took his Homburg hat and his folded umbrella and flew to Germany to come to terms with Hitler, nine out of ten of us, with an instinctive horror of war, said "Thank God!".

Two years later it was hard to find a single human being who had not, so he said, disagreed with Chamberlain. The "men of Munich" became marked men. The idol had not only turned out to have feet of clay, but, oddly enough, had never, so it seemed, been an idol at all!

On September 3, 1939, we listened, from across the sea, to the broadcast words of a declaration of war from a Prime Minister who saw his efforts in ruin about him. An hour later I was telling the people of Australia that we, too, were at war.

To most people the story of Neville Chamberlain came to be a story of ignorance of danger, of unawareness of Hitler's true character, of simplicity confronted by guile, of weak and uncertain action, of ultimate failure. Did Chamberlain, then, contribute nothing to ultimate victory?

The answer is that he gave us time, even at the price of humiliation. There had been, under the air administration of Philip Swinton, a concentration upon quality in the Spitfire and the Hurricane; great "shadow" factories had been set up and equipped. The Battle for Britain had already been partly won. Let us remember that it was won not only by the superb dash, individuality and courage of the pilots, but also by the superior speed, manœuvrability and fire-power of their aircraft.

If Chamberlain really believed that the risk of war was ended at Munich, and if all efforts at armament then slackened, Munich was an unqualified disaster and Chamberlain must be condemned. But I have never quite believed this. True, Germany obtained the Skoda works and other great resources by the rape of Czechoslovakia. But Chamberlain inherited (except on the naval side and the development of fighter aircraft) a largely undefended nation. A year was worth a good deal. We were much stronger in 1939 than in 1938. And, apart from all this, the twelve months after Munich, with their grim and hateful record of treachery and aggression, did much to marshal the decent moral opinion of the world, to harden the spirit of resistance to tyranny and crime.

That is why I believe that the historian will say that Neville Chamberlain, in spite of his undoubted disposition to appease, to seek to solve the problem by postponing it, made his contribution to ultimate victory.

And then the great blows fell and disaster was in the air, and Churchill, who in the opinion of his critics had been, up to that time, always brilliant but mostly wrong, was sent for.

Ramsay Macdonald, Baldwin, Chamberlain, could never have stood in the imminent deadly breach and rallied the forces of freedom against all odds and all reason. That task was for one who understood danger and despised it, whose motto was "action, action, action!", who went down through the poetry and pride of his people into those elemental deeps of courage and defiance and sacrifice and cheerful fortitude which turned aside all attacks.

No English-speaking man or woman of our time will ever forget the thrill of hearing from time to time, over the radio, the voice of Churchill, and of getting from that distant voice a new fire and a renewed bravery.

I will never forget how this great warrior-statesman would enter the historic Cabinet room in Downing Street, take his seat in dead silence, pull his truculent and tilted cigar from his mouth and say:

> Gentlemen, we have the signal honour of being responsible for the government of our country at a time of deadly danger. We will proceed with the business.

It sounds prosaic, as repeated now, but it made our hearts beat the faster. It is hard to believe that there was ever a war leader like him.

But no man could be a great leader without a great people. He evoked and stimulated courage; he did not create it. He himself was and is an unrivalled benefactor to posterity. But those who went before him, with all their faults, made their own contribution to victory. Ultimate justice demands that we should occasionally remember it.

And, of course, there are others of whom I will speak only as I have known them.

Lloyd George was, of course, for all practical purposes a retired and elder statesman when I first met him twenty years ago. But even in 1941 I went down to his farm at Churt and had a full day with him—to me one of intense joy. His silver mane blowing in the wind, his brilliant and penetrating eye, his personal charm and his mastery of language were all, even then, quite irresistible.

I am sure that his distinguished son, the Right Honourable Gwilym Lloyd George, now Home Secretary in the United Kingdom Cabinet, will not mind if I tell you a simple story which illustrates the whole matter.

> Gwilym in 1941 had invited me to lunch at one of the University Clubs in London, together with a couple of other men.
>
> He said to me: "I believe you have been seeing something of my father."
>
> I said: "Yes, indeed I have."
>
> "What do you make of him?" said Gwilym, with a twinkle in the eye.
>
> "Well", I said, "in the last five or six years I don't think he has made a single public speech in the House of Commons or outside it with which I would feel able to agree. Yet, after half an hour with him, if he said to me 'Menzies, I want you to abandon everything that you are doing and follow me', I think I probably would!"

Perhaps the right way to put this matter is to say that the two great crisis leaders of our time have been Lloyd George and Churchill, and that each of them had a magnetic quality possessing almost physical force which drew

men to them and enabled them to attain their most remarkable achievements.

One of Winston Churchill's older contemporaries is Lord Halifax, a former Viceroy of India, a notable Foreign Secretary, and British Ambassador to the United States when I passed through there in 1941. Halifax is a kind of man who can perhaps be produced only in his own country—but not for export. A tall man of rather sombre appearance, deeply religious and scholarly in ecclesiastical matters, he was nevertheless—or because of that fact—one who brought to international relations a dignity, a clarity of mind, an innate sense of justice, which impressed the whole of his contemporaries and sustained on the highest level the greatest traditions of English public life.

A younger contemporary is Lord Salisbury, formerly Lord Cranborne, known to a host of his friends as "Bobbity" Salisbury. This may seem to you to be a strange pseudonym for one who has claims (which he does not make for himself) to be one of the wisest men of our time, but it arose in a simple way. His famous grandfather was Robert; in the next generation there was another Robert, which inevitably became "Bob"—a name which I trust you all treat with suitable respect—and therefore, in the third generation, some distinction had to be made and "Bob" became "Bobbity".

There used to be a somewhat cynical saying that "there are three generations from shirt sleeves to shirt sleeves", or as I believe they used to say in Lancashire, "from clogs to clogs". The whole point of the saying is that it is seldom that genius, or even high talent, will be transmitted for very long. It is therefore a stimulating thing to recall that when the first Queen Elizabeth came to the Throne, a horseman went out through the mire and slush from London to Hatfield to tell the Cecil of those days that there was a new Queen. Only a few years ago, on better roads and by more modern transport, a messenger went out from London to tell the Cecil of these days—Robert Salisbury—that the second Elizabeth had come to the Throne. There is something magnificent and enduring in the Cecil blood.

The present holder of the family title has modesty, good sense, good judgement, high character, imagination and a sense of responsibility so completely blended in him that I would think my life well spent if I had known only him among the great contemporaries of Churchill.

Before I conclude, you might perhaps allow me to refer to two of Winston's great American contemporaries.

The first, of course, was Franklin Delano Roosevelt. It is quite likely that the historians will say that in his declining health he was deceived at Yalta and at Potsdam. Perhaps he was, for his mind was friendly and generous and, towards the end, not easily prepared for cunning or indirect motives.

Of his immense personal charm I can speak from conviction and experience. His courage was enormous. There can have been few men in the history of the world who came through a long and crippling disease ultimately to sit in one of the most powerful places of authority in the world.

It is not for people like me to attempt to estimate his ultimate place in history or the final measure of his intellectual parts. But there can be no doubt that in the very best sense of the word he was one of the master politicians of this century. He knew his own people. He spoke intimately and easily across the wireless to his own people. He was always on our side in the war, but he knew better than anybody else how to handle his own public opinion so that his own great nation would at least not be against us, would certainly at least be the most helpful of neutrals, and would in due course be with us to the end.

His successor, Mr. Truman, was written down when he became President. He appeared to the superficial onlooker to be just a man who had become President by the accident of Roosevelt's death. He answered most of his critics when, single-handed and against all the odds, he retained the Presidency in 1948; but there will remain a perhaps vagrant idea that he was what the Americans would call a "run-of-mine" politician who had not the personality or command of his more famous predecessor.

Well, I have had the opportunity of seeing a good deal of Mr. Truman and of thinking a good deal about what, when he was President, he was called upon to decide and to do. I think, therefore, that I should tell you, without any presumption I hope, that he was and is a great man with qualities of the most essential and remarkable kind. He had many bitter decisions to take, including the crucial decision about the atomic bomb. He took his decisions and never swerved from them. And when his decisions had been taken and the political attacks followed and many newspapers assailed him, he stood to his guns, quite serenely, cheerfully, humanly. I do not think I ever met a more naturally friendly man. I do not think that I have met many men who behind their naturalness and friendliness possessed such pertinacity of mind, such determination to pursue the course seen to be the right one. I would venture to say that any man who possesses decision, courage and endurance has great claims to honour in a world in which time-serving and being all things to all men are so frequently regarded as the marks of a superior political intelligence.

I have named only a few of Winston Churchill's contemporaries. I could have spoken to you for another two hours about 20 or 30 more. But I have mentioned those whom I have named because it is one of my profound beliefs that the greatest men are not lonely accidents, but come out of a generation of great men who provide at once their stimulus and their foil.

In any great man's heyday it is fashionable to eulogize him to excess. When his day has gone, it is, I fear, fashionable to decry him and to get some clever young man to write a book to explain that he was never great at all. The whole purpose of my speech to you tonight has been to endeavour to restore the balance. It will be a poor day for our race when any generation arises which is not able to say with a full heart and true mind: "Let us now praise famous men."

THE HISTORY OF SCURVY IN THE NAVY.[1]

By ERIC SUSMAN AND D. J. DELLER.
From the Royal Prince Alfred Hospital, Sydney.

IN preparing this brief historical fragment, the authors had no little difficulty in establishing a spatial fourth-dimensional relationship between some of the landmarks in the study of scurvy and contemporaneous world events. To help us achieve this object, we drew up the following table (Table I).

We place it at the beginning of this article with the hope that the reader will likewise be aided in getting his historical bearings.

In 1797, a certain Earl Spencer, the first Sea Lord, made an official visit to the Naval Establishments at Portsmouth. With gracious condescension, this nobleman "expressed a humane desire to see the pitiful victims of scurvy" at the neighbouring Naval Hospital at Haslar. He was told that there were no such cases, and you could have knocked His Lordship over with a feather. And it was true. For practical purposes, the disease had vanished overnight.

Starting, then, from this *fin de siècle* incident, we propose to utilize, in the modern technique of the cinema, a "flash-back" of the disease during the eighteenth century. This century saw the disease at its zenith and saw its decline and virtual disappearance, and will serve as a cross section or sample of the whole subject.

[1] Read at a combined meeting of the Section of History of Medicine and the Section of Naval, Military and Air Force Medicine and Surgery, Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August, 1955.

Scurvy was probably unknown to sailors before the sixteenth century. Primitive navigational methods forced ships to hug the coastline, and there was insufficient stowage space for victuals. For these two reasons it was obligatory to put into port for fresh supplies of food and drink. The invention and utilization of the mariner's compass ushered in the era of naval exploration, and the great oceans of the world were soon opened up to an intense and highly competitive maritime traffic.

"Land scurvy" had been known for centuries. It was associated with wars, pestilences, famine, and the days of besieged and beleaguered cities, but this "sea scurvy" was a new disease—a complete novelty.

Notions on the pathogenesis of the malady went through the stages of the usual mediæval pattern, being highly coloured with superstition and religious dogmas. Its irregular and capricious appearances were put down to the baleful operations of the Devil, a recurrent calamity, by Divine permission, as a chastisement for the sins of the world. Pox or syphilis from the north, scurvy from the south, communicated and intermingled, and produced a

TABLE I.

A Chronological Table of Important Eighteenth Century "Scurvy Events".	A Comparative Table of Certain Historical Events to Establish a Temporal Relationship with Scurvy.
1716 James Lind born.	1715 Jacobite rebellion in Scotland.
1740 Lord Anson starts his disastrous world voyage.	1740 War of the Austrian Succession. General European war.
1747 Lind at sea as surgeon in His Majesty's ship *Salisbury*.	1748 Treaty of Aix-la-Chapelle. End of European war.
1753 James Lind's "A Treatise of the Scurvy" published.	1751 Gray wrote his "Elegy in a Country Churchyard".
1758 Lind appointed Physician to Haslar Hospital.	1756 Black Hole of Calcutta. 1757 Battle of Plassey—Clive in India. 1759 Capture of Quebec (Wolfe defeats Montcalm).
1772 Captain James Cook's voyage round the world without a death from scurvy or typhus.	1776 American Declaration of Independence.
1794 James Lind died.	1794. French Revolution. Reign of Terror in Paris.
1796 Routine use of lemon juice ordered in the Royal Navy.	1796 Austria crushed. Bonaparte invades Italy. 1798 Jenner popularizes vaccination.

combined poison—a kind of pathological symbiosis. Then there was the scorbutic taint; a patient inherited the scorbutic diathesis from his parents. There was also the contagious theory; a scorbutic grandfather could, by kissing the infant, convey scurvy to his grandchild. Meteorological concepts were not wanting. The inclement English "fog", the damp dank air from the swamps, and exposure of the body to cold and wet were all thought to play an ætiological role. The position was much the same as it is today with carcinogenesis—a tangled mass of confused, formless nebulous thinking of all theory and no facts. This, then, was the conglomerate hotch-potch of a disease which physicians could not cure or alleviate by orthodox apothecary, when the hero of the scurvy saga, James Lind, staged his entry.

Scurvy *sine* Lind is "Hamlet" *minus* the Prince, so it is fitting that we discourse briefly on this inspired doctor, the father of nautical medicine. Little is known about his early days. Born in 1716, he commenced his apprenticeship to an Edinburgh surgeon at the age of fifteen years. Systematic teaching of medicine had just begun in Edinburgh, and he probably attended professorial classes. The Royal Infirmary was not yet opened for clinical instruction to medical students. At the age of twenty-three years he finished his studies, but took no degree. Tiring of the climatic, dietetic and social vexations and disabilities of his native land, he followed the standard drill of all enterprising Scotsmen. He went south. He joined the Royal Navy as a civilian, with the rating of surgeon's mate. Eight years later, by effluxion of time, he underwent a curious metamorphosis, and from sick berth attendant he became a surgeon, and was drafted to His Majesty's Ship *Salisbury*. It was on this voyage that he made his monumental contributions to the study of the disease. A year later he retired from the Navy, and took his degree of doctor of medicine at Edinburgh. In 1753 the "Treatise" was published—one of the great medical monographs of all time. In 1758 Lind left Edinburgh for Portsmouth to take over the new naval establishment, Haslar Hospital, at that

FIGURE I.

Observe the alert, interested scholarly expression. Remark a fleeting suggestion of sardonic humour, and a paradoxical element of playfulness and whimsy, together with a fastidious detachment from the ordinary and conventional. But, overall, a warm, kindly facies.

time the largest hospital in Europe. In his first two years of office there were 5734 admissions. One in five of these men was suffering from scurvy. The Channel Fleet might put into Portsmouth and land 1000 men incapacitated by the malady. Naval surgeons of the day had come to dread the critical period of ten weeks at sea, which was the "warming-up" or "incubation period" of this all too familiar scourge. Lind's *Salisbury* observations were fortified and extended by an enormous experience of the disease during his Haslar superintendency.

Lind retired from Haslar after a quarter of a century's devoted service, and died two years later. Two years after his death the Admiralty ordered the routine use of lemon juice. There was a hideous lapse of forty-two years between that "Papal Bull" and the publication of the "Treatise". So much for the man. Now for his work. Even to the most superficial and casual reader, the "Treatise" has all the earmarks and stamp of genius. Lind took nothing for granted. He was prepared to stand or fall by his careful

observations and the precise recording of the facts. He had a profound contempt for the voluminous and misleading literature of his time—an evil under which every member of this Congress labours today. He was essentially a man of his century—the age of reason. As an example of his rational thinking, he despised all the contemporaneous notions on witchcraft.

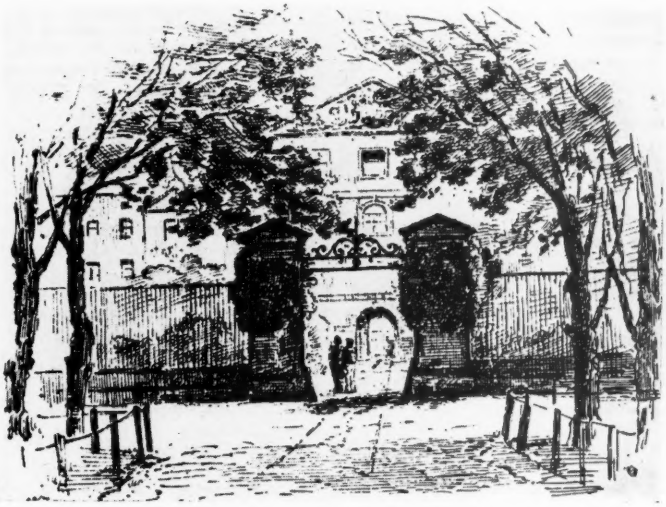


FIGURE II.
Entrance to Haslar Hospital.

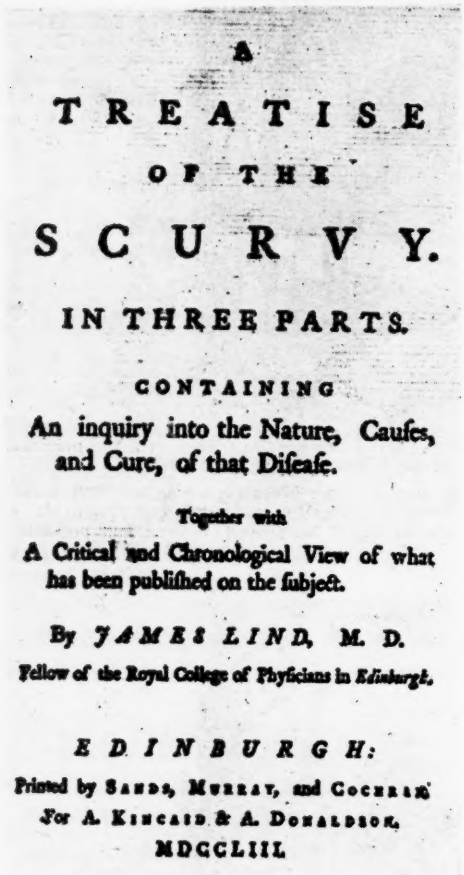
He showed quite clearly that scurvy was unknown in the ward room, and that it flourished amongst the lazy and the indolent—skulkers, as they are known in the Navy. He could anticipate what we now call the "stress factors" in disease, and observed its frequency in the poor victims of press gang recruitment. "Those that are of a chearful and contented disposition are less liable to it than others of a discontented and melancholy mind." We can imagine him calling on Hans Selye, in Canada, and, within the hour, having acquired the new vocabulary (or jargon, if you prefer it), getting on famously with the professor on a plane of psychosomatic equality.

Lind, so far as we are aware, was the first investigator to conduct a perfectly controlled experiment in clinical research, in which he proved, beyond any possible doubt, that the juice of citrus fruit was vastly superior to all other forms of reputedly efficacious treatment of scurvy. His post-mortem studies on scorbutic corpses were models of clarity and observational discipline. He did not fail to

FIGURE III.
Euridyce Monument, Haslar Cemetery: "And the sea gave up the dead."
In those days this was a very busy department of the hospital.

recognize the sterno-chondral subluxation so characteristic of the surviving remnant of this disease as we see it in modern times—infantile scurvy or Barlow's *Krankheit*. He drew attention to the havoc which scurvy created in the Navy, and pointed out that shipwreck, prisonership and enemy action were, by comparison, mere trivialities. He cited, by way of example, Lord Anson's disastrous voyage, and quoted the graphic descriptions of the disease during this tragedy by the clergyman Richard Walter.

His clinical picture of florid scurvy is a masterpiece of descriptive writing. He stressed the early symptoms of preternatural fatigue, the weakness and lassitude, the gingivitis, the multiple hæmorrhagic phenomena, and the



A

TREATISE

OF THE

SCURVY.

IN THREE PARTS.

CONTAINING

An inquiry into the Nature, Cauſes, and Cure, of that Diſeaſe.

Together with

A Critical and Chronological View of what has been publiſhed on the ſubject.

By JAMES LIND, M. D.

Fellow of the Royal College of Phyſicians in Edinburgh.

EDINBURGH:

Printed by SANDS, MURRAY, and COCHRAN

For A. KINCAID & A. DONALDSON.

MDCCLIII.

FIGURE IV.
Title page of the "Treatise".

delayed healing of wounds. It would be a grave impertinence to paraphrase this grand passage on a scorbutic mirage.

> In such a situation the ignorant sailor and the learned physician will equally long, with the most craving anxiety, for green vegetables and the fresh fruits of the earth . . . . And such people in the height of the malady not only employ their thoughts all day long on satisfying this importunate demand of nature, but are apt to have their deluded fancies tantalised in sleep with the agreeable ideas of feasting upon them on land. What nature, from an inward feeling, makes them thus strongly desire, constant experience confirms to be the most certain preventive and best cure of their disease.

Freud, 150 years later, writing in the supercharged atmosphere of the Viennese suburbs, could not have expressed this "slumbery agitation" with greater imagery or with deeper feeling.

Lind, as we all know, did not claim originality for his discovery; but by a clinical experiment he converted a suspicion into a certainty—a suspicion that had lurked and flitted at the back of men's minds for two long centuries. Stereotyped ideas and notions on most subjects die hard; medicine is not exempt from this generalization. It was difficult even for the nimble brain of the eighteenth century physician to envisage a negative cause of disease—a lack, an absence, a "something missing" concept.

Ignorance, and a dislike of mankind for change or innovation, are still with us. Sir Sheldon Dudley, sometime Medical Director-General of the Navy, relates that, even as recently as 1942, he was trying to explain the meaning of the phrase "hygiene-discipline" to a high-up admiral. No names, no pack drill. In his best quarter-deck fashion, this dumb-cluck retorted: "What's all this fuss about malaria? After all, it's only a doctor's racket." *Sancta simplicitas*!

And so we return to our Point of Departure, 1797, one year after the introduction of a citrus subsidy, and only eight short years before Trafalgar. James Lind and his pupils and successors, Blane and Trotter, had done a mighty good job. We, in this generation, have known what it is to live under the threat of potential tyranny and dictatorship. So did Englishmen at the dawn of the nineteenth century. God is not only on the side of the big battalions, but on the side of the good doctors. Surely it is very gratifying to us here, in 1955, to reflect that one of our own brethren can, in Elysium, stand side by side with Horatio Nelson, and maintain, in his modest, unassuming manner, that British medicine played a grand and glorious part in protecting the "precious jewel, set in the silver sea" from the fulfilment of the sinister and dreadful designs of Napoleonic ambition.

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EPIDEMIC HYPERPYREXIAL HEAT STROKE.

By A. T. PEARSON,
Perth.

DEATH from hyperpyrexial heat stroke has not been recorded frequently in Australia, and it has often been stated, sometimes in the law courts, that death does not occur from this condition in this country. As far as can be ascertained, the only articles appearing in the Australian medical literature are those of K. G. Hearne (1932) and of N. B. Friend (1932). Cilento (1942) makes the following statement:

> Heat hyperpyrexia is rare to very rare, and heat exhaustion of clinical degree occurs, as elsewhere during heat waves, particularly among old people, alcoholics and the chronically sick.

The Perth Epidemic.

During February, 1955, Perth experienced most unusual and disastrous weather conditions, which led to the death of at least 14 people from hyperpyrexial heat stroke; in all but one case the diagnosis was confirmed by autopsy. At the same time other affected people recovered, and many cases of heat exhaustion and heat cramp were recorded at Royal Perth Hospital and other metropolitan hospitals.

The Weather.

From February 14 to 17, 1955, over six inches of rain were recorded in Perth as against an average monthly total of 38 points. Commencing on February 19, the tem-

perature rose rapidly and with it the humidity, and the heat wave lasted until February 28. The wet and dry bulb temperatures are shown in Table I, with the number of deaths each day.

In the production of hyperpyrexial heat stroke in this epidemic, the wet bulb maximum temperatures are probably the most significant. It is considered that life is not compatible with a wet bulb maximum of 85° F., or over. In Perth, a wet bulb temperature of 80° F. is rare, having been previously recorded only nine times, but never on three successive days as was the case on February 19, 20 and 21. The average maximum wet bulb temperature for February is 68·9° F. In the opinion of the Weather Bureau, the temperatures were probably higher in many parts of Perth, in view of the fact that the observatory is on approximately the highest point in the city. There is evidence that hyperpyrexial heat stroke can also be produced by a heat wave of long-continued high temperatures with very low humidity, as was recorded by Hearne.

TABLE I.

Date (1955).	Wet Bulb Temperatures. (Degrees Fahrenheit.)		Dry Bulb Temperatures. (Degrees Fahrenheit.)		Deaths.
	Maximum.	Minimum.	Maximum.	Minimum.	
February 19 ..	80·0	68·4	95·2	70·9	Nil
February 20 ..	81·8	74·7	103·1	80·0	Nil
February 21 ..	80·2	74·8	105·0	83·3	6
February 22 ..	77·8	72·0	94·1	78·2	2
February 23 ..	78·6	66·0	95·2	70·6	1
February 24 ..	78·8	68·8	94·4	70·0	Nil
February 25 ..	79·8	70·2	97·7	76·9	3
February 26 ..	76·0	65·1	93·1	70·9	2
February 27 ..	76·9	64·6	98·4	70·0	Nil
February 28 ..	76·9	69·8	100·4	75·6	Nil

Autopsy Findings.

Autopsies were performed by myself on 12 male subjects during the last week in February, and some details are given of these cases in Table II. One other case came to autopsy at the Royal Perth Hospital.

The significant findings at autopsy, present in all cases, were as follows.

1. Post-mortem staining was dark purple and extensive, with showers of black petechial hæmorrhages on the skin, especially around the back of the neck and shoulders.

2. The lungs were heavy, dark blue and wet. Section showed the cut surface to be almost black, and gross pulmonary œdema to be present.

3. Showers of petechial hæmorrhages were found on the surface of the heart, especially in the region of the base.

4. There was acute venous congestion of the abdominal viscera.

5. The bladder was empty.

6. No other obvious cause of death was found.

Symptoms and Signs.

Since none of the subjects were examined by me before autopsy, reliance had to be placed on information obtained by the attending medical practitioner or even the police officer who attended, since all were coroner's cases.

Generally, the onset and progress of symptoms were rapid. Sometimes there was complaint of feeling very hot, with headache, giddiness and weakness. These symptoms were quickly followed by restlessness, delirium, collapse and coma.

The body temperature readings were obtained in only six cases and were as follows: 111°, 108·4°, 108°, 106° and 104° F. In Case VI the temperature was 95° F. on the patient's admission to hospital, rising to 100·2° F. before death.

During the heat wave many other patients were treated at the Royal Perth Hospital for the effects of heat. Of these, there were four suffering from heat cramp, three from heat exhaustion and three from heat stroke who recovered. The following is the history and progress of one of the patients who was treated for hyperpyrexial heat stroke and recovered.

A male patient, aged seventy-two years, was admitted to hospital on February 22 at 5.50 p.m., unconscious, and with a mouth temperature of 107° F.; his pulse rate was 120 per minute. His skin was hot and dry, his axillæ were damp

TABLE II.

Case Number.	Age. (Years.)	Sex.	Circumstances Related to Death.	Highest Body Temperature Recorded. (Degrees Fahrenheit.)	State of Skin.	Other Findings.
I	78	M.	Collapsed at home; lived five hours; comatose; twitching of muscles.	104·0	Not recorded.	Blood pressure, 90 millimetres of mercury, systolic, 70 millimetres, diastolic; heart disease present; malnourished and dehydrated.
II	51	M.	Collapsed at work; lived one hour; comatose.	111·0	Hot and dry.	Pulse rate, 120 per minute; pupils fixed; rales and rhonchi in chest.
III	23	M.	Collapsed at mental hospital; died rapidly.	108·0	Not recorded.	Nil.
IV	62	M.	Collapsed at mental hospital.	108·4	Not recorded.	Obese; large heart; coronary atheroma; large fatty liver.
V	66	M.	Collapsed after return from work; died rapidly.	106·0	Hot and dry.	Delirious; small pupils; heavy drinker.
VI	56	M.	Collapsed at work; legs gave way and he fell; legs felt numb; lived 12 hours.	95·0 Rose to 100·2 before death.	Perspiring and cold.	Very dehydrated; pulse not palpable; respiration rate, 44 per minute.
VII	66	M.	Found dead in small, airless room.	Not recorded.	Dry and hot.	Heart disease present; alcoholic.
VIII	36	M.	Collapsed in bathroom; died rapidly; onset of *rigor mortis* very rapid.	Not recorded.	Hot and dry.	Complained of headache; spent restless night; alcoholic.
IX	64	M.	Collapsed at work; rapid death.	Not recorded.	Hot and perspiring.	Obese; very cyanosed.
X	76	M.	Felt ill; went to bed; died three hours later.	Not recorded.	Not recorded.	Heart disease present.
XI	57	M.	Collapsed at mental hospital.	Not recorded.	Not recorded.	
XII	65	M.	Found on floor gasping; hot room; dressed in flannels.	Not recorded.	Not recorded.	Obese; delirious; heart disease present; enlarged liver.

and his liver was enlarged. Neck stiffness and a generalized maculo-papular rash were present. The pupils were small and did not react to light. A diagnosis of pontine hæmorrhage was made. He was placed in bed with a wet sheet over him, with an electric fan blowing on the sheet. Ice packs were placed in the axillæ. At 6.50 p.m. the mouth temperature was 103° F., and he had regained consciousness. At 9.15 p.m. his temperature was 101° F. and pulse rate was 84 per minute. He made a dramatic recovery, and was discharged from the hospital fully recovered. On February 23 his blood chemistry was as follows: the urea content was 43 milligrammes per 100 millilitres, the sodium content was 126 milliequivalents per litre and the potassium content was 3·6 milliequivalents per litre.

Treatment.

In hyperpyrexial heat stroke the treatment depends upon a rapid assessment of the patient, and the immediate application of measures to lower the body temperature before irreparable damage is done to the vital centres of the brain stem. The measures adopted in the case of recovery described above are recommended. Hearne found that the most effective method was to strip the patient, direct a flow of air from an electric fan on to the body and at the same time spray the patient with water. Other measures such as replacement of fluid and salt should follow as soon as possible.

Comment.

After seeing the number of cases and deaths which occurred in the February heat wave in Perth, I am rather of the opinion that hyperpyrexial heat stroke must not be as rare as was previously thought, especially in tropical and subtropical Australia. Since it can occur during the course of ordinary work, it would appear to be a compensable condition. Hearne, after a lifelong study of heat stroke, also holds this view.

A significant feature of the fatal cases in this series was that most were incorrectly diagnosed. Pontine hæmorrhage was diagnosed four times on the signs of hyperpyrexia, collapse, convulsions and coma. Of cerebral hæmorrhages, pontine hæmorrhage is an uncommon type, and it is possibly less common, in view of the fact that hyperpyrexial heat stroke can simulate it. No statistics are available in this State concerning the percentage of cerebral hæmorrhages which occur into the *pons cerebri*.

Summary.

1. An epidemic of hyperpyrexial heat stroke occurring in Perth in February, 1955, is described, with at least 14 deaths.

2. The associated abnormal temperatures are tabulated.

3. The clinical conditions and autopsy findings are recorded.

4. Differential diagnosis and treatment are discussed.

Acknowledgements.

I wish to thank the Weather Bureau for their invaluable assistance with details of the temperatures, and the Medical Superintendent of the Royal Perth Hospital, for permission to report the cases which occurred in his hospital.

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RECENT EXPERIENCES WITH STAPHYLOCOCCAL INFECTION IN CHILDHOOD.

By L. I. TAFT,

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LATE in the year 1953 the occurrence, over a short period, of a striking number of deaths from staphylococcal infection led to a review of the disease for a period of twelve months. There were 41 deaths from staphylococcal infections, and the clinical histories and pathological findings in selected cases are presented below.

The increasing incidence and severity of infection began in June, and cases continued to occur until December. The incidence of these infections is indicated in Table I; the mortality and types of cases are classified in Table II.

Thirty-seven patients with acute respiratory infection yielding positive cultures were admitted to the hospital during June-July, 1953. The incidence of isolation of the staphylococcus from these patients is shown in Table III.

Dubos, in his text-book, states that 9% of cases of bronchopneumonia are due to the staphylococcus, the mortality being high. In this selected group the incidence is very much higher. Of 39 children who died of pneumonia, in 1953, staphylococcal infection was a significant factor in 37 instances.

Although the use of antibiotics has resulted in the widespread appearance of resistant forms of staphylococci, the frequency and severity of these infections are such that this is not likely to be the explanation, and suggest the possibility of virulent strains.

The 41 deaths referred to in Table II may be classified in three main groups, as follows: (i) apparent primary staphylococcal infections, even though in most of these cases some debilitating factor was also present; (ii) a group of cases of fibrocystic disease of the pancreas; (iii) terminal infections occurring in patients with other debilitating incurable diseases. Thirteen case histories are presented to illustrate the features of severe staphylococcal infections in infants and children; in these there are 11 examples of primary staphylococcal infection, including two non-fatal cases, and two cases of fibrocystic disease of the pancreas.

Reports of Cases.

CASE I.—A., a male baby, aged seven months, developed a croupy cough two days prior to his admission to hospital. Laryngeal stridor and chest retraction were present on the morning of his admission, but his condition deteriorated rapidly and he died in the ambulance on the way to hospital. At necropsy the vocal cords were œdematous and covered by greyish membrane, which virtually closed the glottis. The false cords presented a similar appearance. The tracheal mucosa was congested, and the bronchi contained tenacious mucopus extending down into the smaller bronchi. Both lungs were œdematous. Microscopic examination of the epiglottis revealed congestion, œdema and leucocytic infiltration. *Staphylococcus pyogenes* was recovered from the trachea.

CASE II.—B., a male baby, aged three and a half months, had a "bad cold" with cough for four days prior to his admission to hospital, with dyspnœa and gross circulatory failure of some hours' duration. On clinical examination of the child the heart was slightly enlarged, the lungs were clear and the liver could be palpated three fingers' breadth below the costal margin. A provisional diagnosis of cardiac failure was made. Digitalis was administered and oxygen was given, but the baby expired twelve hours after his admission to hospital. At necropsy suppurative bronchitis was present, culture of material obtained from a bronchial swab producing a profuse growth of *Staph. pyogenes*. Mild enteritis and slight cardiac dilatation were also present, but there was no microscopic evidence of myocardial damage. Attempted culture of microorganisms from blood obtained *post mortem* gave negative results. The circulatory failure was presumed to be toxic in origin.

CASE III.—C., aged eighteen months, a well-nourished child, presented with a history of "a cold" of four days' duration. During the previous day he had developed a cough,

TABLE I.

The Incidence of Staphylococcal Infections as Reflected in Laboratory Cultures, June-July, 1953.

Specimen.	Number of Specimens Received from which a Pathogen was Recovered.	*Staphylococcus Pyogenes.*	β-Hæmolytic Streptococcus.	*Streptococcus Pneumoniæ.*	*Hæmophilus Influenzæ.*	*Corynebacterium Diphtheriæ.*	Gram-Negative Bacilli.[1]
Throat swabs	142	56	68	14	3	1	—
Nasal swabs	7	7	—	—	—	—	—
Ear swabs	25	13	4	1	3	—	4
Conjunctival swabs ..	8	4	3	—	—	—	1
Wound swabs	72	44	8	—	—	—	20
Pus from bone	9[2]	8	1	—	—	—	—

[1] Includes *B. coli communis*, *Pseudomonas pyocyanea* and *Proteus vulgaris*.

[2] For the whole of 1953. Three positive blood cultures of *Staph. pyogenes* were obtained from these cases.

dyspnœa and vomiting. He was cyanosed, with rapid grunting respirations and signs of consolidation at the base of the left lung. He was given oxygen, penicillin, streptomycin and chlortetracycline, but on the day after his admission to hospital he died suddenly.

At necropsy extensive consolidation of both lungs was found, the left being gangrenous. More than 300 millilitres of serosanguineous fluid were present in the left pleural cavity, which was lined by a fibrinous exudate. Thin pus was present in the bronchi. Microscopic examination of a section from the base of the left lung revealed extensive necrosis and hæmorrhage into the parenchyma, with leucocytic infiltration and commencing breakdown of lung tissue. At the pleural surface many colonies of staphylococci could be seen beneath the fibrinous exudate (Figures IV and V).

TABLE II.

Mortality Figures for Staphylococcal Infections, 1953.[1]

Type of Infection.	Number of Cases.
Primary respiratory tract infection:	
Tracheobronchitis	1
Bronchitis	1
Pneumonia	8 } 20
Pneumonia and empyema	8
Pneumonia and pyopneumothorax	2
Pneumonia with congenital heart disease	2
Tonsillectomy complicated by staphylococcal *enterocolitis*	1
Fibrocystic disease of the pancreas, staphylococcal bronchopneumonia	9
Terminal pneumonia occurring in moribund patients	9

[1] Total number of post-mortem examinations, 208; total number of deaths associated with proven significant staphylococcal infection, 41.

CASE IV.—D., a premature baby, was discharged from a maternity unit aged twelve days, weighing five pounds nine ounces. Feeding difficulties were initially encountered, and four days prior to his admission to hospital his mother was thought to have influenza. He was admitted to hospital at the age of three weeks with a twenty-four hours' history of anorexia, vomiting and drowsiness. On his admission to hospital his temperature was 98° F., his pulse rate was 110 per minute, and his respirations numbered 30 per minute. Crepitations were audible over both lung fields. Despite treatment with warmth, oxygen, penicillin and streptomycin, his condition deteriorated over several hours. Just prior to death he coughed up blood-stained sputum.

At necropsy plum-coloured discoloration of the trachea was found, becoming more intense at the bifurcation and along the main bronchi. Examination of the cut surfaces of the lung revealed uniform consolidation except at the apices and anterior margins of both upper lobes. The air passages were lined by intensely congested mucosa with profuse catarrhal exudate.

Culture of material from a bronchus produced a profuse growth of a penicillin-resistant *Staph. pyogenes*. Attempted isolation of virus from lung tissue was unsuccessful because of bacterial contamination.

Microscopic examination of sections of the lung revealed fibrinous exudate, red cells and a few small round cells in the air passages. There were visible staphylococcal colonies in the exudate. The superficial layer of ciliated columnar epithelium in the larger bronchi was desquamated. The alveoli, many of which were emphysematous, contained exudate and red cells. The maternal history of influenza and the tracheobronchitis with desquamation of epithelium, suggest that this was primarily a virus infection of the influenzal type, complicated by a secondary staphylococcal infection.

CASE V.—E., aged two weeks, was stated to have had snuffles and a nocturnal cough since birth. He had rapid

TABLE III.

Incidence of Staphylococci in 37 Cases of Respiratory Tract Infection, June-July, 1953.

Pathogen Isolated.	Number of Cases.
Staphylococcus pyogenes	27
β-hæmolytic streptococcus	4
Pneumococcus	3
Bacillus coli communis	3

grunting respirations for one day prior to his admission to hospital. On examination, he was seen to be a sick baby, with a temperature of 102·4° F. and a pulse rate of 130 per minute; his respirations numbered 34 per minute. Crepitations were audible at the base of the right lung. Mild umbilical sepsis was present. Radiological examination of the chest revealed an extensive opaque area in the right lung, with displacement of the heart to the left, and depression of the liver. *Staph. pyogenes* sensitive to chlortetracycline was recovered from a cough swab. This drug was given after initial chemotherapy with penicillin and streptomycin. On the day after his admission to hospital some thick pus was aspirated from the right pleural cavity, but subsequently no gross pus collection could be localized. The baby remained ill for some days and erythromycin was given, but then general deterioration with cyanosis and peripheral circulatory failure occurred. Chest aspiration produced only a few millilitres of blood-stained pus, and the baby died after a profuse hæmoptysis some minutes later.

At the post-mortem examination the trachea and bronchi were not inflamed, but contained a large blood clot extending down into both lungs. Examination of the left lung revealed a little patchy consolidation, and the pleural cavity was clear. The upper lobe of the right lung was collapsed, and the lower lobes were the site of extensive confluent abscesses, into some of which hæmorrhage had occurred. The visceral and parietal layers of pleura were lined by a thick layer of fibrino-purulent material extending over the right hemidiaphragm, and in the loculations of the pleural cavity approximately 25 millilitres of blood-stained pus were present. Macroscopic examination revealed confluence of multiple small abscesses in a section of the middle lobe of the right lung. Pulmonary hæmorrhage and consolidation were present in some areas, and in other portions interstitial œdema,

alveolar collapse and infiltration with small round cells were found. Large mononuclear cells were present in the alveolar spaces of congested areas. The bronchi and bronchioles contained pus and blood. In most of these the bronchial epithelium was intact.

CASE VI.—F., a male baby, aged nine months, was adopted from a babies' home, having just recovered from a respiratory tract infection. He still had a cough. Four days prior to his admission to hospital he became apathetic, and three days later had rapid grunting respirations. Examination revealed him to be pale, cyanotic and moribund; his temperature was 103° F., his pulse rate was 200 per minute, and his respirations numbered 60 per minute and were shallow and gasping in quality. The percussion note was dull at the base of the

FIGURE III.

Case VI. Post-mortem specimen of lung showing multiple abscesses, one of which has ruptured onto the pleural surface resulting in pyopneumothorax.

right lung. Radiological examination of the chest revealed right lower lobar consolidation with presence of some pleural fluid. *Paracentesis thoracis* resulted in the withdrawal of one millilitre of thick pus, from which *Staph. pyogenes* was recovered. Over the next week the baby was given oxygen, penicillin, streptomycin and chlortetracycline, with improvement, but purulent sputum persisted. Nine days after his admission to hospital he suddenly became shocked. Radiological examination of the chest confirmed the presence of a right-sided pneumothorax (Figure I[1]). On paracentesis air was released under positive pressure, and four millilitres of thick blood-stained pus were removed. The administration of oxytetracycline was commenced, but the baby's condition remained poor over the next day. Further radiological examination again revealed a tension pneumothorax, which was needled with some improvement in the baby's condition. Three hours later he suddenly collapsed and died.

At necropsy the trachea and bronchi contained thick yellow pus. The right lung was the site of numerous abscesses, containing thick yellow pus surrounded by areas of consolidation. Several subpleural abscesses were present, two of which had ruptured into the pleural cavity. The left lung was emphysematous, with basal consolidation. Microscopic examination confirmed the presence of confluent pulmonary suppuration. Gross fat accumulation was present in the liver.

[1] In the legend to Figure I on the special plate a reference is made to Figure VIII. This should read Figure III.

CASE VII.—G., a baby, aged three weeks, developed paronychia of the left thumb which failed to respond to penicillin therapy. Multiple subcuticular abscesses on the left thumb were incised. Culture resulted in the isolation of *Staph. pyogenes*, which was sensitive only to chlortetracycline. Radiological examination revealed no evidence of osteomyelitis. Chlortetracycline was given, but despite this, infection spread, and the baby developed multiple necrotic skin lesions with central purulent collections. Despite the intravenous use of oxytetracycline and blood transfusions for anæmia, the lesions spread, and perianal cellulitis supervened followed by gangrene. The baby died from pneumonia and peripheral circulatory failure when aged six weeks. Examination of sections of perianal skin revealed extensive necrosis of epithelium, subcutaneous fat and muscle. No evidence of moniliasis was present.

CASE VIII.—H., a male child, aged three years and six months, was admitted to hospital with a history of cough, fever and dyspnœa of six days' duration. There had been no response to penicillin, streptomycin and chlortetracycline therapy. Radiological examination of the chest demonstrated the presence of an extensive cyst containing air and fluid, in the dorsal area of the right lung extending into the apex. There was consolidation of the base of the right lung, and slight mediastinal deviation to the left. A similar cyst was present at the base of the right lung. Mantoux and Casoni tests produced negative results. *Staph. pyogenes*, sensitive to chlortetracycline and insensitive to penicillin and streptomycin, was isolated from a cough swab. Chlortetracycline therapy was continued with little improvement. Three days later fluoroscopic examination of the chest revealed an apparent decrease in the fluid in the large cyst, and smaller cystic areas were visible, with some consolidation and pleural thickening in the base of the lower lobe of the right lung (Figure II). Oxytetracycline therapy was substituted, with improvement, which was then interrupted by a bout of measles. Radiological examination revealed clearing of the base of the right lung, which disclosed numerous small cystic spaces. The large cyst was practically unchanged. Oxytetracycline therapy was discontinued, but three days later the patient's temperature rose. Radiological examination revealed thickening around the large cystic space in the right lung. Oxytetracycline was recommenced, and with continued physiotherapy he improved. He was discharged to a convalescent home with treatment to continue for one month, after which he was given "Distaquaine" penicillin daily for three weeks. Posturing produced a little yellow sputum at this stage, and radiological examination now revealed considerable resolution. A small cystic area in the right dorsal zone and some segmental collapse in the right anterior basic segment remained. Follow-up examination since has revealed complete clearing of the right lung.

CASE IX.—I. was a full-term baby weighing six pounds nine ounces, who had been delivered instrumentally. He suffered from a facial paresis. When aged two weeks he developed a breast abscess which responded to treatment. Two weeks prior to his admission to hospital he developed a preauricular abscess, for which he was given penicillin. This discharged into the right external auditory meatus, with considerable reduction in the facial paresis. When he was aged six weeks he was admitted to hospital with cough, pallor and rapid grunting respirations. On examination, the baby was seen to be jaundiced and apathetic; his temperature was 90° F., his pulse rate was over 200 per minute, and his respirations numbered approximately 100 per minute. The throat was reddened, and right otorrhœa was present. Fine rales were audible over the left side of the chest, and the liver was palpable six centimetres below the right costal margin. Some neck stiffness was present. Lumbar puncture revealed opalescent cerebro-spinal fluid containing 340 polymorphonuclear cells, 25 lymphocytes and 150 red cells per cubic millimetre; no organisms were seen in the smear. *Staph. pyogenes* was recovered from a culture of the cerebro-spinal fluid, the blood and the right auditory meatus. The organism was penicillin-resistant, but sensitive to chlortetracycline, oxytetracycline and erythromycin. Blood examination indicated anæmia and neutrophilia. A radiological examination of the chest demonstrated an extensive opaque area and cystic change in the left lung, while in the right lung there was an enlarged hilar shadow, with numerous opaque areas throughout the lung tissue. These findings prompted the diagnosis of staphylococcal pyæmia, for which penicillin, erythromycin and chlortetracycline (intravenously) were given. During the illness abscesses appeared under the tongue and on the left little finger. These required incision. An episode of acute rise in intracranial pressure with

cyanosis and opisthotonos occurred, and later dye tests indicated poor absorption of cerebro-spinal fluid, suggesting the occurrence of intracranial venous thrombosis. Anæmia was counteracted by blood transfusions. Repeated lumbar punctures showed clearing of the cerebro-spinal fluid. Over a period of two months the cystic appearance in the lung

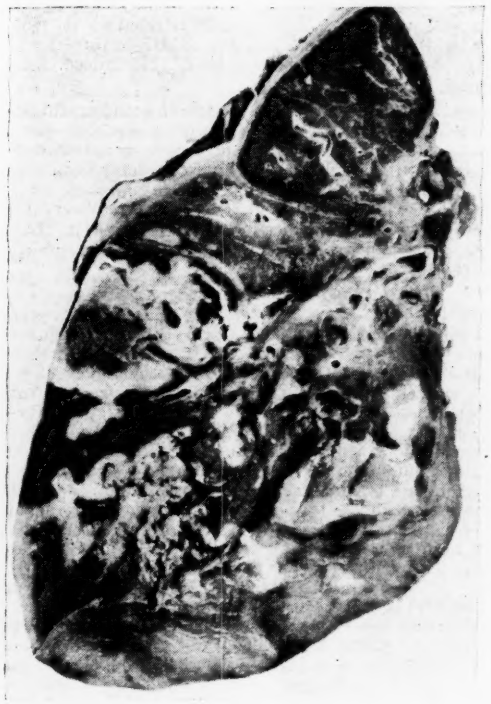

FIGURE VI.

Case XII. Post-mortem specimen of lung from a patient with staphylococcal pneumonia, showing infarct, multiple abscesses and fibrinous pleurisy.

resolved, leaving some opacity in the upper lobe of the left lung, with generalized increase in markings on the right side. Two months after the child's discharge from hospital there were clinical and radiological signs of sclerosing osteitis of the ramus of the right mandible. Pus was aspirated from this region, culture resulting in the isolation of *Staph. pyogenes*. A small sequestrum was later discharged through a sinus which developed after the aspiration of pus. This sinus subsequently healed.

CASE X.—J., a male child, aged seven years, diagnosed as suffering from fibrocystic disease of the pancreas, died from suppurative bronchopneumonia. *Staph. pyogenes* was repeatedly isolated from his sputum. Repeated courses of chlortetracycline during the twelve months prior to death had failed to alleviate his pulmonary infection.

At necropsy the trachea and main bronchi were found to contain a large quantity of greenish-yellow pus. The walls of the dilated bronchi were inflamed and thickened and full of pus. The lungs were grossly emphysematous, with patchy areas of consolidation.

Microscopic examination of lung sections revealed hyperplastic respiratory epithelium in the larger air passages, but peripherally there were complete loss of epithelium, gross thickening of the bronchiolar walls by granulation and fibrous tissue and loss of smooth muscle. Examination of the alveoli revealed patchy areas of collapse, consolidation and emphysema. Chronic inflammatory changes were present in the hilar glands.

The diagnosis of fibrocystic disease of the pancreas was confirmed histologically.

CASE XI.—K., a female baby, aged one month, was admitted to hospital with a history of failure to thrive, frequent profuse stools, and the recent onset of a harsh cough. On examination, the baby was emaciated, and respirations were rapid, with inspiratory chest retraction. *Staph. pyogenes* was isolated from a cough swab. Radiological examination of the chest revealed emphysematous lung fields, with increased markings, and segmental collapse of the upper lobe of the right lung. Fat globules were present in the stools, and no tryptic activity was detected.

The baby was regarded as suffering from fibrocystic disease of the pancreas, and died of bronchopneumonia two weeks after her admission to hospital. At necropsy the diagnosis was confirmed. Suppurative bronchopneumonia was present. Both lungs were very emphysematous and the subpleural lymphatics of the upper lobe of the left lung were distended with air. Numerous air-containing cysts were present.

On microscopic examination the bronchi were found to contain purulent exudate, and were lined by hyperplastic epithelial cells distended with mucus. Patchy bronchopneumonic changes were visible. The cysts were lined by alveolar epithelium and interstitial tissue (Figures VII and VIII).

CASE XII.—L., a female baby, aged eleven days, had been delivered normally after a normal pregnancy. She contracted a mild upper respiratory tract infection at the maternity hospital. Three days after her discharge from hospital she developed a cough, vomiting and rapid respirations.

FIGURE VII.

Case XI. Post-mortem specimen of left lung from a patient with fibrocystic disease of the pancreas and staphylococcal pneumonia, showing emphysema, and multiple cysts in the upper lobe.

She was dyspnœic, and had clinical and radiological signs of collapse of the lower lobe of the left lung and consolidation of the upper lobe. Emphysema and pneumonitis were present on the right side. Despite chlortetracycline therapy, her condition progressively deteriorated and death occurred twenty hours after her admission to hospital. *Staph. pyogenes* sensitive to chlortetracycline, and *B. coli* resistant to chlortetracycline were isolated from a cough swab.

At necropsy there was minimal inflammation of the trachea and large bronchi. The left lung was consolidated, and the lower dorsal area of its upper lobe was very dark and appeared to be infarcted. Small multiple abscesses were present throughout the remainder of this lung, being most obvious in the lower lobe. One of these had ruptured into the left pleural cavity. This cavity contained blood-stained serous fluid. A thick layer of fibrin covered the pleura of the lower lobe of the left lung. In the upper lobe of the right lung several areas of suppurative consolidation were present. Some flakes of pus were present in the pericardial cavity, and both layers of pericardium were inflamed

Microscopic examination of sections of lung revealed a diffuse suppurative process, with disappearance of the alveolar structure, areas of hæmorrhage and necrosis. Many colonies of staphylococci were visible. The interstitial tissue was œdematous and infiltrated with polymorphonuclear cells, but the bronchial epithelium was generally intact. Fibrinopurulent exudate was present on the pleura.

This case illustrates the rapid, extensive and severe inflammatory lung damage produced by the organism. Only collapse and emphysema had been evident radiologically twenty hours before death; yet abscess formation, empyema, pulmonary infarction and pericarditis were present at necropsy.

CASE XIII.—M., a female child, aged three years and six months, underwent a tonsillectomy for repeated attacks of *otitis media*. On the fourth day after operation she began to vomit severely and frequently. The vomiting was soon followed by the onset of diarrhœa; this lasted for one day, during which she passed approximately twenty profuse watery stools. On her admission to hospital she presented the picture of severe peripheral circulatory failure and dehydration. Intravenous therapy was only temporarily effective, and the child died within two days of her admission to hospital. At necropsy severe entero-colitis was found, with extensive mucosal necrosis. A pure growth of *Staph. pyogenes* was obtained from a swab of the depths of a washed ulcerated patch of mucosa. Microscopic examination of sections revealed colonies of staphylococci in the wall of the bowel. No other pathogen was obtained from stool culture.

It is a matter of speculation whether the naso-phayngeal wound contributed to the ingestion of the organism. The tonsillar fossæ were apparently healing normally during the illness.

Pathological Features.

The basic changes found in the tissues as a result of staphylococcal infection were necrosis and pyogenic inflammation. Necrosis was found in relation to clumps of staphylococci amounting to microscopic colonies (Case III, Figure IV). Here necrosis was presumably due to the direct action of staphylococcal toxins. The colonies and the tissue damage in lesions only one or two days old bore witness to the rapid multiplication of the organisms and to the severity of the infection. In the lungs some extensive areas of necrosis were seen whose peripheral distribution and macroscopic and microscopic appearances left no doubt that these were infarcts, presumably the result of vascular involvement in the inflam matory process (Case XII, Figure V).

Modifications of these basic changes occurred with the site and severity of the infection and the inflammatory response in the patient. In Case VII the cutaneous lesions were predominantly necrotic, with little cellular reaction. Severe mucosal infection resulted in pseudomembranous (Case I) and ulcerative (Case XIII) lesions. Thus a cholera-like illness with profuse diarrhœa and peripheral circulatory failure resulted from enteric infection. In the respiratory tract of the infant the relatively narrow air passages were particularly prone to obstruction by the products of inflammation. Here the clinical manifestations ranged from the inspiratory stridor and costal retraction of laryngo-tracheo-bronchitis (Case I) to the emphysema and tension air cysts of bronchiolitis (Cases VIII, IX and XI, Figures II, VI and VII). The pulmonary effects of bronchiolitis were particularly evident in cases of fibrocystic disease of the pancreas (Cases X and XI). Segmental collapse was common, but massive collapse was usually associated with intrapleural collections, emphysema, air cysts *et cetera* (Case XII).

Within the lung itself, all grades of inflammation from congestion and hæmorrhage to grey hepatization were seen. Areas of necrosis proceeded to abscess formation. These abscesses were usually small, multiple, and distributed in the peripheral areas of the lung (Case V). Not uncommonly rupture of a subpleural abscess (Case VI) resulted in pyopneumothorax, sometimes of the tension type (Figures I and III). Pleural inflammation often occurred early in the disease, serofibrinous pleurisy proceeding to empyema formation. The pericardium was similarly involved in Case XII.

Toxæmia was often prominent, manifested by fever, leucocytosis and peripheral circulatory failure, while in Case II the patient presented with clinical evidence of myocarditis. Histological evidence of enteritis was also present in this case. Some diarrhœa was common in these infants, but the relative part played by direct infection and staphylococcal enterotoxin is unknown. The significance of staphylococci isolated from the fæces of children with an otherwise non-specific enteritis is not yet clear, while severe diarrhœa due to direct infection (Case XIII) has not commonly been recognized at this hospital.

Anæmia (Case VII) and hepatic fat infiltration (Case VI) were observed in patients who survived for some weeks. More recently cases of severe renal dysfunction associated with staphylococcal infection have been observed and histological evidence of tubular damage has been obtained. This may be linked with the observation of Trueta, who produced degenerative changes in the renal cortex by the intravenous injection of staphylococcal toxin into rabbits.

Pathogenesis.

The events leading up to the pathological end results in the lungs can be pieced together into a sequence suggesting the natural history of the disease. In some cases there was continuity of the inflammatory process from the larynx caudally, suggesting a descending infection from the naso-pharynx — a known habitat of the staphylococcus. This was seen in fibrocystic disease of the pancreas and in terminal pneumonia. In the former, the larger amount of viscid mucus is said to favour a descending staphylococcal infection, while in the latter, poor respiratory excursion, depressed cough reflex and the collection of secretions likewise facilitate descending infection. In Case IV, one of apparently primary staphylococcal tracheo-bronchitis and bronchopneumonia, there was a descending type of inflammation. This patient presented a history of contact with a person probably suffering from influenza during a current influenza B epidemic in Melbourne (Anderson and McLean, 1953). Histological features of influenzal tracheobronchitis were found—namely, superficial respiratory epithelial degeneration and desquamation (Stuart-Harris, 1953).

Attempted isolation of virus was unsuccessful, however, and serological studies could not be performed. Previous coincidence of influenza with severe staphylococcal pneumonia has been reported by Tyrrell (1952), by Finland *et alii* (1942), and by Burnet, Stone and Anderson (1946), and more recently, a report of the isolation of influenza virus from the respiratory tract of two infants dead of staphylococcal laryngo-tracheo-bronchitis and pneumonia was issued by MacDonald and Brown (1955) and by Derrick (1955).

Verlinde and Maksteinits (1953) showed, in monkeys, that penetration by staphylococci is apparently facilitated by the epithelial lesions and pulmonary œdema produced by the influenza virus. They also showed that staphylococcal infection has an unfavourable influence on the resistance of the epithelium to the virus. Isolation of virus was attempted on post-mortem material in seven cases of this series; but bacterial contamination and the age of the lesions militated against recovery of the virus. Serological investigation in two cases yielded negative results. Since then, during the winter of 1954, an influenza C epidemic in Melbourne was again associated with a large number of cases of staphylococcal pneumonia at the Royal Children's Hospital. It would seem probable that influenza does play a part in a few of these cases, and confirmation by isolation of virus and by serological tests on patients in the ward would be desirable. In other cases the absence of involvement of the larynx, trachea and bronchi, and the miliary distribution of lesions in the lungs and other organs might support the view that the infection was bloodborne from a superficial focus (Case XI), such as paronychia (Case VII) or umbilical sepsis (Case V). This could have been clarified by blood cultures, which, however, were not attempted as a routine procedure.

Epidemiology and Predisposing Factors.

Staph. pyogenes is widely distributed in nature, being present in air, dust, water, food and sewage. In man the primary habitat of the staphylococcus is in the naso-pharynx, the skin being secondarily infected from this source. Droplet infection, direct contact and fomites provide means of spread from one person to another. The air and dust of hospital wards and nurseries have been shown to be heavily contaminated with the organism (Rountree and Barbour, 1950). These workers also demonstrated, by phage typing techniques and sensitivity tests, that in nurseries the staphylococci obtained from the babies' naso-pharynx reflected the strains prevalent in the nursery environment and staff, rather than those from maternal sources.

Cunliffe (1949) found that in a maternity unit over 90% of infants became infected in the first two weeks of life. A significant decrease in this figure was found in infants born at home. He further demonstrated a fall in the nasal carrier rate after the age of one month to approximately 23%, this probably being related to the isolation of the infant in the small closed home environment, and the development of immunity to the prevalent organisms in that environment. The carrier rate then increased to approximately 64% during the school years and slowly fell, to become stabilized at the adult level of 30% to 60%.

Bryce and Burnet (1932) showed that at birth circulating staphylococcal antitoxin levels were high, being derived passively from maternal sources; they fell rapidly in a few weeks, to be slowly built up during childhood years. This fact, with the severe spreading lesions of the infants, the high carrier rate of infants in hospitals, yet the low incidence of staphylococcal lesions amongst mothers of neonates with severe infections, suggests that the nasal mucosa and skin of the infants are peculiarly susceptible to infection by the staphylococcus.

Ten of the 20 patients with primary staphylococcal infection were admitted to hospital directly from maternity units, or within several days of discharge from maternity units, or from a foundling home. Furthermore, 16 of 20 patients who died of pneumonia were aged less than six months. It is remarkable also that the severest lesions with a tendency to disseminate locally and metastatically occurred in the early months of life.

Debilitating states were present in most of the fatal cases of primary staphylococcal pneumonia. Failure to thrive was present in five cases, hypothermia in five cases, prematurity in two cases, and parental neglect in one case.

Sensitivity of Staphylococci to Antibiotics.

Review of the sensitivities of the strains of staphylococci isolated during this period has paralleled the findings of Forbes (1949) and of Finland and Haight (1953)—namely, that the incidence of resistance to antibiotics is related to the exposure of the organism to the respective antibiotics. Thus, of 34 strains of staphylococci isolated immediately on the patient's admission to hospital during June and July, 1953, there was a higher proportion of sensitive strains to current antibiotics compared with 31 strains recovered from patients who had been in hospital for more than one week. Patients from the second group were admitted from maternity units or foundling homes, were close contacts with recently discharged in-patients, or had already been subjected to chemotherapy. The introduction of newer antibiotics to deal with resistant organisms has merely postponed the problem. Thus the widespread use of chlortetracycline to treat staphylococcal infections in this hospital during the latter half of 1953 was associated with a drop in the percentage of sensitive strains from 90·6 to 74·0.

A review of the sensitivities of the staphylococci recovered from 28 fatal infections revealed that only one strain was sensitive to penicillin and 11 strains were sensitive to streptomycin. The incidence of resistant strains was significantly higher among those isolated from 11 patients who developed their terminal infection in hospital. Of 17 patients admitted to hospital with an ultimately fatal staphylococcal infection, 13 empirically received an in-vitro effective antibiotic coverage, but six died from toxæmia and pulmonary damage within forty-eight hours of their admission. Only four may have been saved by the use of other antibiotics. It seems that the fulminating nature of the infection is such that few more patients would be saved by currently used antibiotics, while in other cases tissue damage prevents the establishment of effective local concentrations of antibiotics. Progression of suppuration results in empyema or pyopneumothorax which may be fatal. Further information regarding the nature of the staphylococci is being sought by phage typing through the courtesy of Dr. Phyllis Rountree.

Summary and Conclusions.

1. Mortality figures at the Royal Children's Hospital, Melbourne, during 1953 disclosed the high incidence of deaths associated with infections by *Staph. pyogenes*. During the period June–July, 1953, a survey of cultures revealed a high morbidity from infections by this organism.

2. Representative case histories are presented, the clinical course and pathological findings encountered indicating the fulminating nature of the illness, which was associated with severe toxæmia, tissue necrosis and suppuration.

3. Evidence of both hæmatogenous dissemination from superficial foci of infection and tracheo-bronchial spread is presented. In one case an initial influenzal infection is suspected.

4. Hospital cross-infection with terminal pneumonia and cases of fibrocystic disease of the pancreas contributed significantly to the mortality from staphylococcal infection.

5. Antibiotic-resistant staphylococci are progressively increasing, particularly since the large-scale use of antibiotics in treating these infections. Their occurrence in hospitals, maternity units and foundling homes, from which a significant number of fatal cases originated, further emphasizes the dangers of cross-infection. The use of penicillin and streptomycin alone as initial chemotherapy in suspected staphylococcal infections in babies would no longer appear to be rational.

6. Death occurs from toxæmia and pulmonary damage in many cases before antibiotics can have any effect. Tissue necrosis and suppuration prevent the establishment of an effective local concentration of antibiotics, and pleural involvement from rupture of an abscess may cause death at a later stage.

7. These problems have arisen with an organism which served originally as a measure of the potency of penicillin. They are not being permanently solved by the newer antibiotics.

Acknowledgements.

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Reports of Cases.

ADAMANTINOMA.

By ERIC M. FISHER,
Sydney.

DURING the development of the enamel organ in the embryo, a structure like a cap is formed which consists of the following parts: (i) the external enamel epithelium towards the surface of the mouth; (ii) an internal enamel epithelium which, in contact with the dental papilla, will

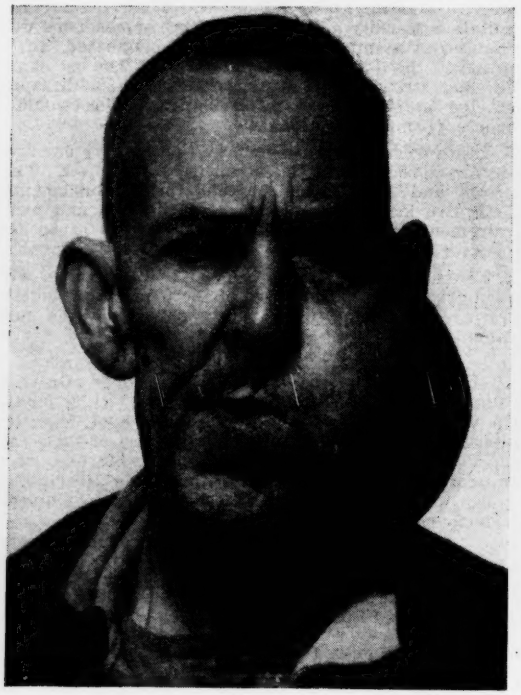

FIGURE I.
Case I: patient before operation.

produce enamel, and this is the only part of the enamel organ which does so; (iii) a relatively thick zone between the two, consisting of epithelial cells connected by long processes and separated by intercellular fluid and known as the stellate reticulum.

The external layer and the stellate reticulum will form the periodontal membrane around the fully developed tooth. Epithelial residues of the dental lamina persist about the developing tooth, and from these arise the so-called adamantinoma and the intraalveolar carcinoma of the jaw.

Certain parts of an adamantinoma resemble the stellate reticulum, and the typical epithelium is found in clumps resembling basal-celled carcinoma, or may resemble very closely a squamous carcinoma with cornification.

These tumours do not form any enamel, but may contain unerupted teeth. They may seem to be formed of large numbers of small or large cysts as the intercellular spaces become filled with fluid.

Willis (1953) describes the tumour as follows:

> Adamantinoma is a lowly malignant slowly growing epithelial tumour of rather characteristic microscopical structure which closely resembles that of the developing enamel organ but which does not form enamel.

The tumours arise in the substance of the jaw and by their growth invade and destroy it.

They grow slowly, rarely metastasize and unless wholly removed, recur locally. It is common to find that the history includes one or more operations.

Dew and Miller (1932) make the following statement:

> The constant tendency towards local recurrence after partial operation is an index of the truly malignant nature of the lesion and, as is the case with other types of quasi-malignant tumours, each recurrence seems more active than the last.

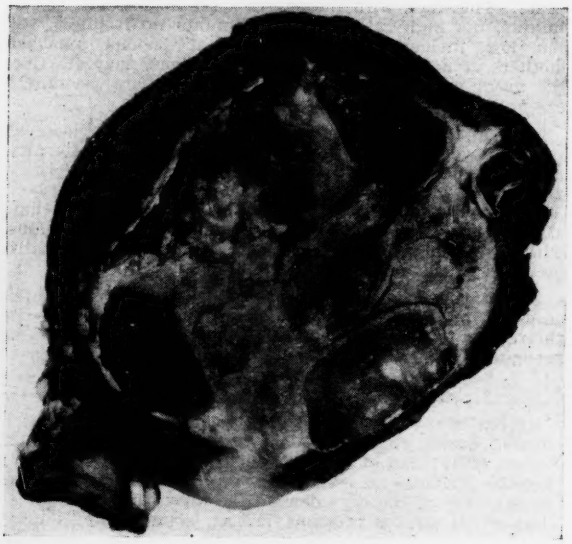

FIGURE II.
Case I: tumour on section.

Bland Sutton (1922) states that the tumours are painless unless they ulcerate; this usually occurs into the mouth, and it is remarkable how little inconvenience they cause.

Adamantinomata occur in the lower jaw in over 80% of cases (Willis, 1953), and grow till they destroy the structure of the bone. Usually the tumour is limited to half the mandible, but it may involve both sides. They project into the mouth sufficiently to interfere with speech and swallowing, and may displace the floor of the mouth and the soft palate as far as the middle line. In the upper jaw they may spread widely and beyond operative limits in the small bones of the face.

In the Royal Prince Alfred Hospital records since 1910, there are 11 cases under the diagnosis of adamantinoma or fibrocystic epithelioma; but it is possible that others are filed under other headings. Three of these 11 tumours were in the upper jaw and eight in the lower. Six were in males

ILLUSTRATIONS TO THE ARTICLE BY L. I. TAFT.

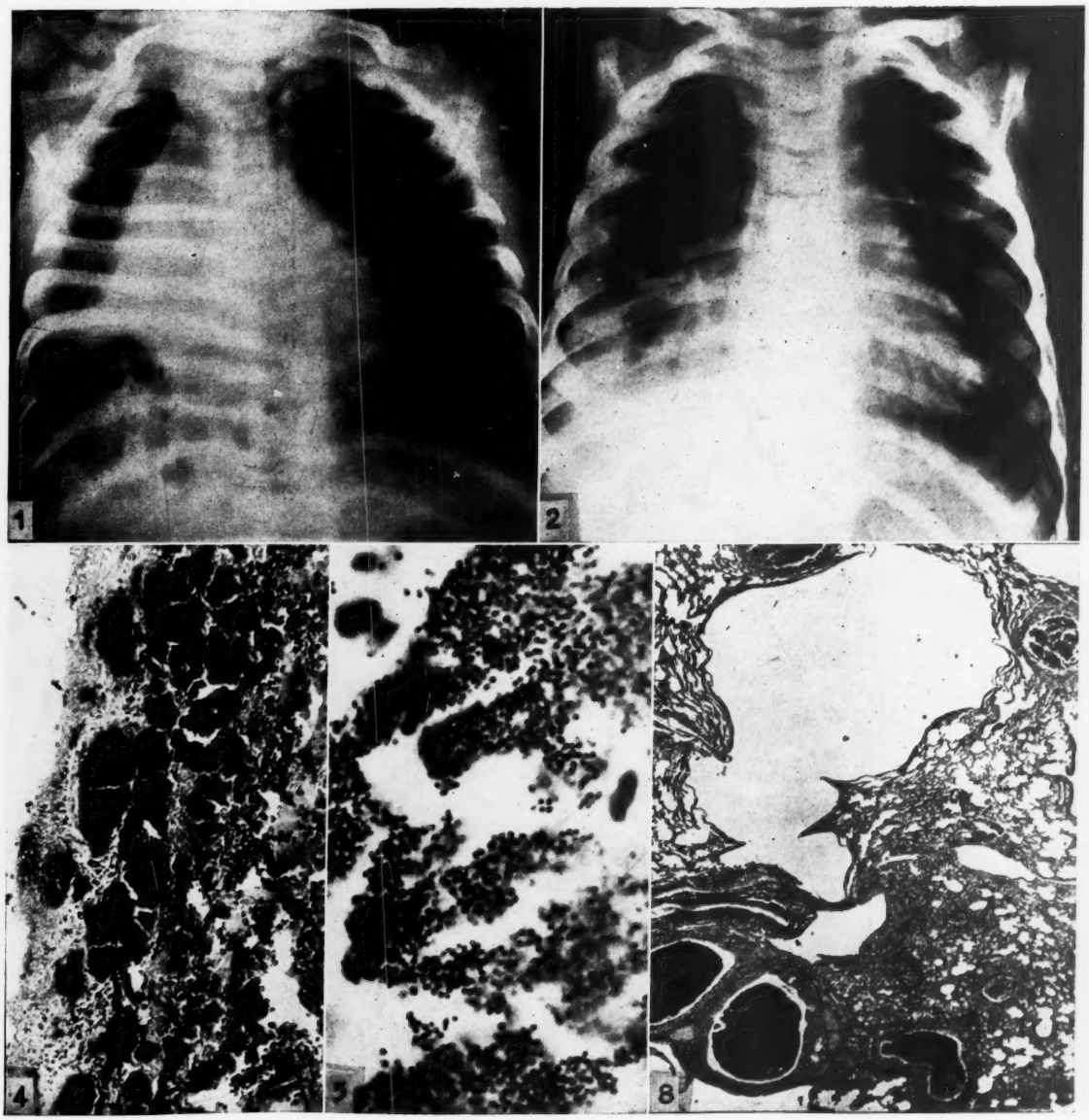

FIGURE I: Case VI, radiograph of chest, showing right-sided pyopneumothorax (see Figure VIII). Figure II: Case VIII, radiograph of chest, showing presence of cysts in the right lung with consolidation at the base. FIGURE IV: Case III, photomicrograph showing pleural surface with multiple "colonies" of staphylococci. (Hæmatoxylin and eosin stain, ×180.) FIGURE V: Case III, photomicrograph showing typical staphylococcal morphology in pleural "colonies". (Gram stain and oil immersion, ×1600.) FIGURE VIII: Case XI, photomicrograph showing emphysema and air cysts associated with obstruction of bronchi and bronchioles. (Hæmatoxylin and eosin stain, ×15.)

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Sydney.

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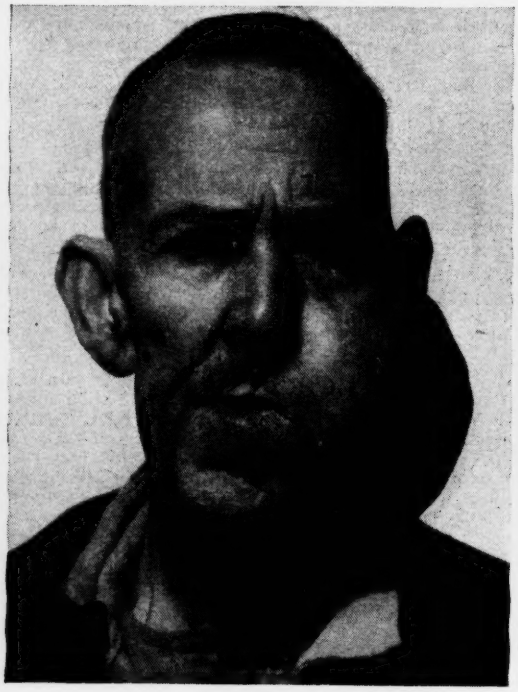

FIGURE I.
Case I: patient before operation.

produce enamel, and this is the only part of the enamel organ which does so; (iii) a relatively thick zone between the two, consisting of epithelial cells connected by long processes and separated by intercellular fluid and known as the stellate reticulum.

The external layer and the stellate reticulum will form the periodontal membrane around the fully developed tooth. Epithelial residues of the dental lamina persist about the developing tooth, and from these arise the so-called adamantinoma and the intraalveolar carcinoma of the jaw.

Certain parts of an adamantinoma resemble the stellate reticulum, and the typical epithelium is found in clumps resembling basal-celled carcinoma, or may resemble very closely a squamous carcinoma with cornification.

These tumours do not form any enamel, but may contain unerupted teeth. They may seem to be formed of large numbers of small or large cysts as the intercellular spaces become filled with fluid.

Willis (1953) describes the tumour as follows:

> Adamantinoma is a lowly malignant slowly growing epithelial tumour of rather characteristic microscopical structure which closely resembles that of the developing enamel organ but which does not form enamel.

The tumours arise in the substance of the jaw and by their growth invade and destroy it.

They grow slowly, rarely metastasize and unless wholly removed, recur locally. It is common to find that the history includes one or more operations.

Dew and Miller (1932) make the following statement:

> The constant tendency towards local recurrence after partial operation is an index of the truly malignant nature of the lesion and, as is the case with other types of quasi-malignant tumours, each recurrence seems more active than the last.

FIGURE II.
Case I: tumour on section.

Bland Sutton (1922) states that the tumours are painless unless they ulcerate; this usually occurs into the mouth, and it is remarkable how little inconvenience they cause.

Adamantinomata occur in the lower jaw in over 80% of cases (Willis, 1953), and grow till they destroy the structure of the bone. Usually the tumour is limited to half the mandible, but it may involve both sides. They project into the mouth sufficiently to interfere with speech and swallowing, and may displace the floor of the mouth and the soft palate as far as the middle line. In the upper jaw they may spread widely and beyond operative limits in the small bones of the face.

In the Royal Prince Alfred Hospital records since 1910, there are 11 cases under the diagnosis of adamantinoma or fibrocystic epithelioma; but it is possible that others are filed under other headings. Three of these 11 tumours were in the upper jaw and eight in the lower. Six were in males

ILLUSTRATIONS TO THE ARTICLE BY L. I. TAFT.

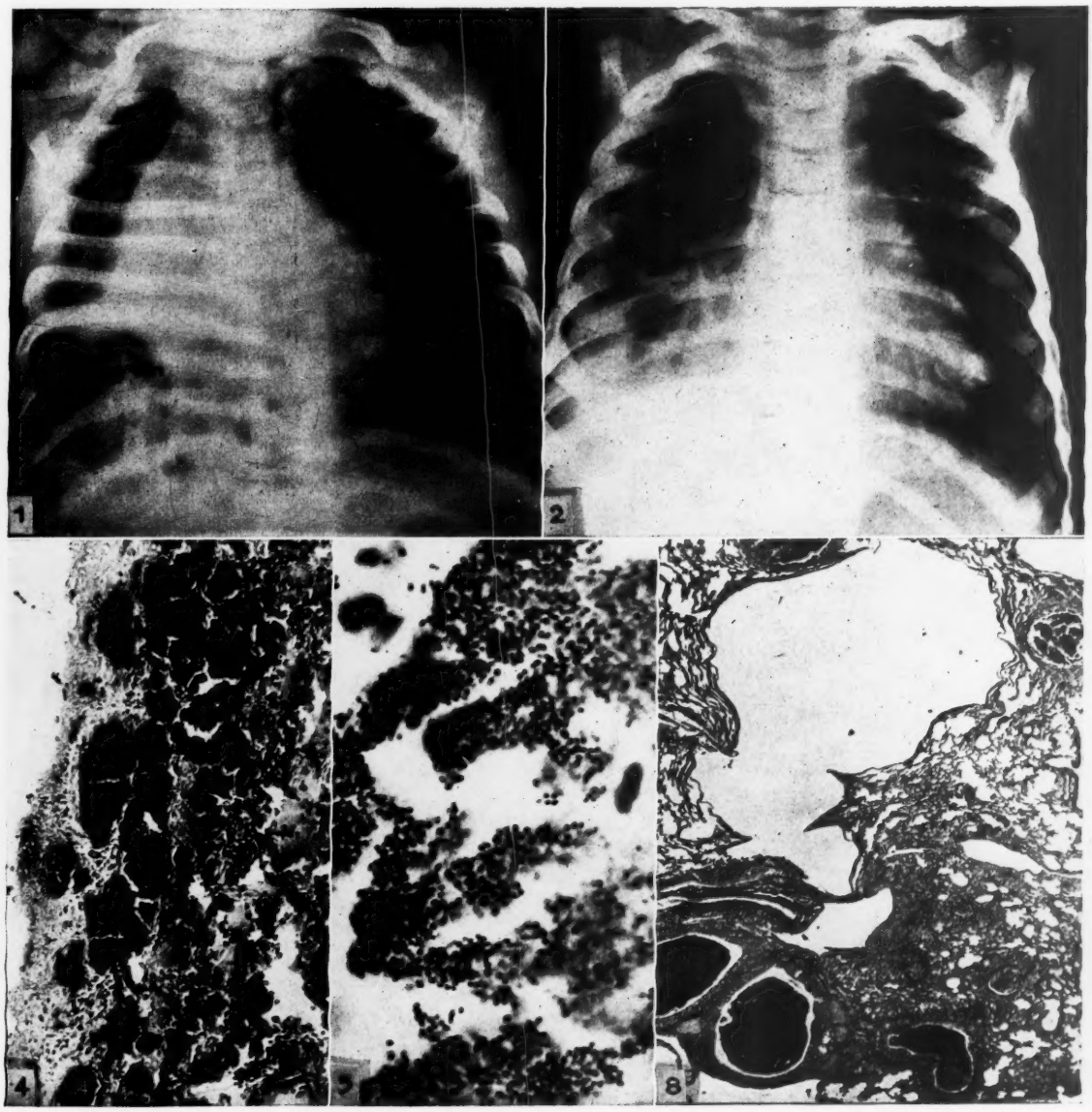

FIGURE I: Case VI, radiograph of chest, showing right-sided pyopneumothorax (see Figure VIII). Figure II: Case VIII, *radiograph of chest, showing presence of cysts in the right lung with consolidation* at the base. FIGURE IV: Case III, photomicrograph showing pleural surface with multiple "colonies" of staphylococci. *(Hæmatoxylin and eosin stain, × 180.)* FIGURE V: Case III, photomicrograph showing typical staphylococcal morphology in pleural "colonies". *(Gram stain and oil immersion, × 1600.)* FIGURE VIII: Case XI, photomicrograph showing emphysema and air cysts associated with obstruction of bronchi and bronchioles. *(Hæmatoxylin and eosin stain, × 15.)*

ILLUSTRATIONS TO THE ARTICLE BY ERIC M. FISHER.

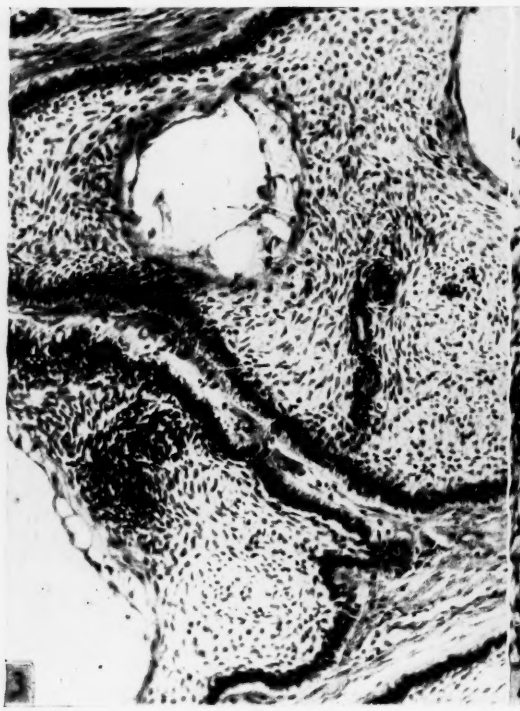

FIGURE III.

FIGURE IV.

ILLUSTRATION TO THE ARTICLE BY KENNETH W. STARR.

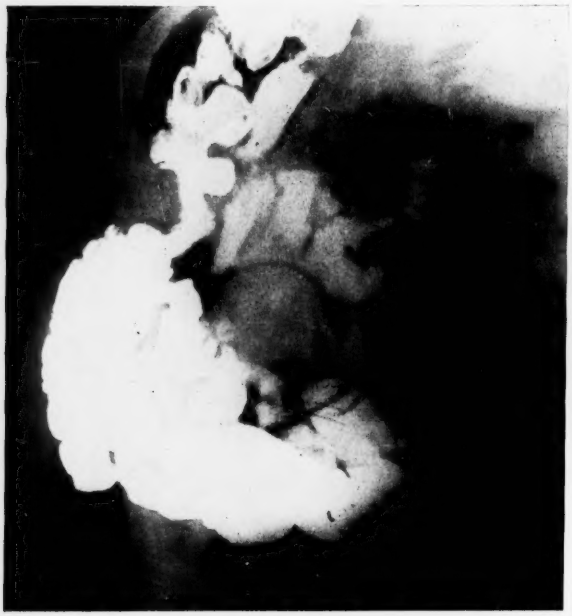

FIGURE II.

ILLUSTRATION TO THE ARTICLE BY THOMAS F. ROSE.

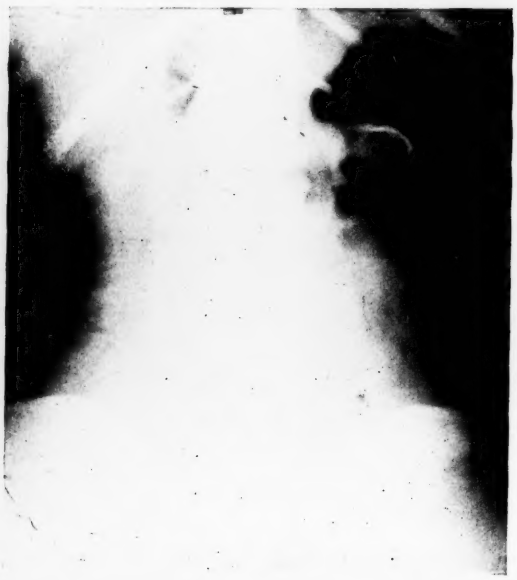

FIGURE I.

and five in females. The youngest patient was a man aged eighteen years, and the oldest a man aged sixty-five years. The following cases are reported.

Case I.

A., a male patient, aged sixty-five years, had a ten years' history of a swelling which began at the angle of the left mandible. It increased slowly, but in the previous four months had grown rapidly. It had been quite painless; but the recent increase in size had caused difficulty in talking and swallowing and deafness in the left ear. He had some dyspnœa on exertion and had lost two stone in weight in the previous two months. He had had deep X-ray therapy without any improvement.

Examination of the patient showed a huge cystic tumour extending from near the symphysis to the external auditory meatus and behind the ear—from the zygoma to well into the neck and to the mid-line inside the mouth. The mucous membrane was thinned in one spot, but not ulcerated.

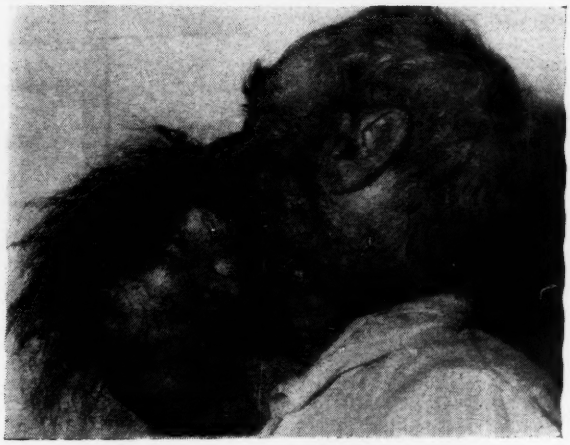

FIGURE V.
Patient in Case II.

There were bony fragments to be felt, but on the whole the tumour was cystic. The overlying skin was stretched, but not adherent. The tumour moved freely when the patient opened and closed his mouth. X-ray examination revealed complete destruction of the left half of the mandible and its replacement by cystic loculated growth which showed areas suggestive of malignant change.

On May 24, 1949, the left half of the mandible was excised. The left external carotid artery was first ligated with some difficulty, as it was overhung by the tumour. The jaw was divided through normal bone just to the left of the symphysis, and the left half of the mandible removed entire without separation of the condyle (which has occurred at times). Dr. G. Davies reported that the tumour was mainly an adamantinoma, but parts of it resembled intraalveolar epidermoid carcinoma.

Convalescence was smooth, and speech and swallowing were quite satisfactory. The deformity was more noticeable than usual, as the excision of the condyle left an obvious depression, and the deep X-ray treatment had rendered the left half of the face hairless.

Case II.

B., aged sixty-five years, a male patient, was admitted to the Prince Henry Hospital in April, 1954. Fourteen years earlier a small, hard swelling had appeared on the right half of the mandible, which in six months became the size of a hen's egg. This was operated on in Brisbane, but recurred in six months. It increased to its present size gradually, and was painless till six months prior to examination, when a hole appeared within the mouth on the right side of the tumour. The patient did not complain of difficulty in swallowing or speaking and was otherwise well.

Examination of the patient revealed a huge tumour, mostly cystic, in which long bars of bone could be felt, and which involved both halves of the mandible, the right side of the tumour being the larger. Both ascending rami were involved, and a large bar of bone could be felt on the left side which seemed to represent the posterior edge of the ramus. There was a large opening in the floor of the mouth on the right side, between the cheek and the tongue, which admitted the tip of a finger. The skin over the lower part of the tumour on the right side was adherent and red and close to ulceration, and there were areas inside the mouth where the mucosa was adherent and thin. The whole tumour moved when the mouth was opened, and could be rocked up and down. The patient had allowed long coarse hairs to grow to hide the deformity, and refused to have them removed for photography.

The condition was considered to be inoperable.

Summary.

Two advanced cases of adamantinoma are reported.

References.

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Legends to Illustrations.

FIGURE III.—Typical adamantinoma, with cystic areas in the stellate reticulum.

FIGURE IV.—Transformation of the stellate reticulum into squamous carcinoma.

MASSIVE TUMOUR OF RIGHT GROIN WITH LARGE TRACTION HERNIA.

By KENNETH W. STARR,
Sydney.

A., A FEMALE PATIENT, aged forty-seven years, had had an increasing swelling in the right groin since childhood. In 1946, during pregnancy, it enlarged considerably; in 1954 she fell and bruised the tumour, which formed an abscess, discharged naturally and healed. She was referred to me shortly after this event by Dr. Charles Salisbury. Apart from the discomfort of its size there were no symptoms associated with it or its contents. The size and appearance of the tumour and the breadth of its pedicle are evident in Figures IA and IB. Clinical and radiological examination revealed that the pedicle contained a large traction hernia with the neck, five inches in diameter, replacing the entire inner inguino-femoral region. The hernia contained large and small gut (Figure II) and omentum, but it was readily reducible. The tumour was soft in consistency and covered by delicate wrinkled skin resembling that of the scrotum. The edge of the defect in the abdominal wall in the inguinal region could be defined; the defect through which the hernia passed was five by five inches in area. Scattered elsewhere over the skin of the body were numerous small, soft tumours, some of them sessile, and a large patch of *café-au-lait* staining was present on the skin of the right side of the abdomen.

Operation was performed under penicillin-streptomycin cover on July 16, 1954. Transfusion was required. The findings were as follows. An enormous tumour was present; it was soft and vascular, and contained a large traction hernia. The hernia was inguino-femoral in nature; there was a deficiency in the inguinal ligament. The defect in the parietes was about five by five inches in area. The

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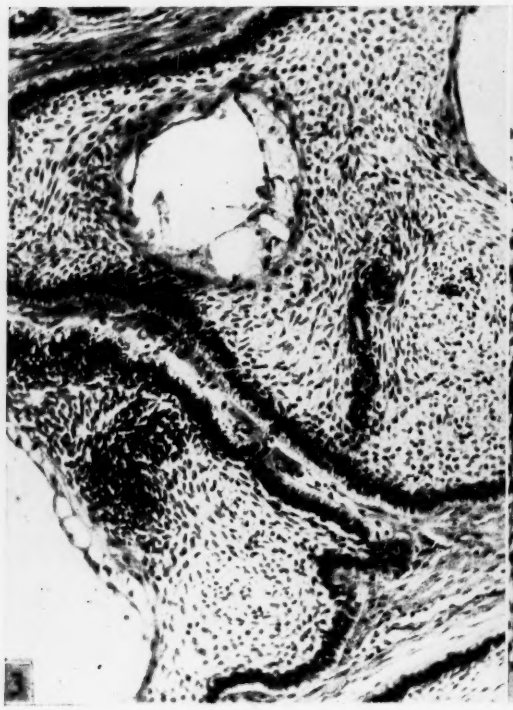

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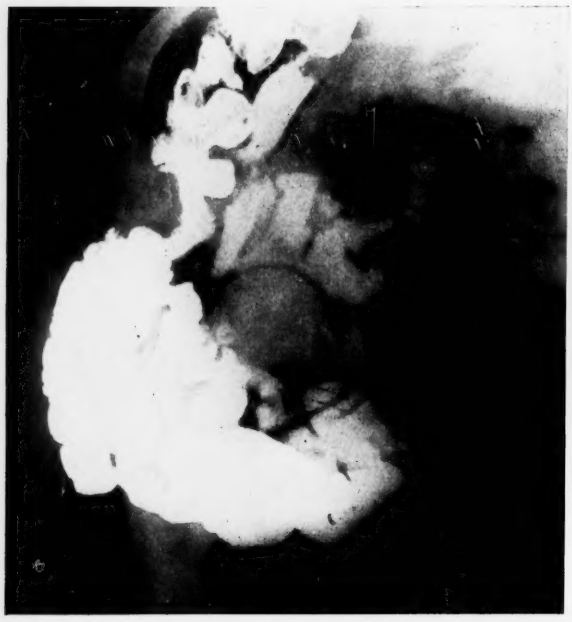

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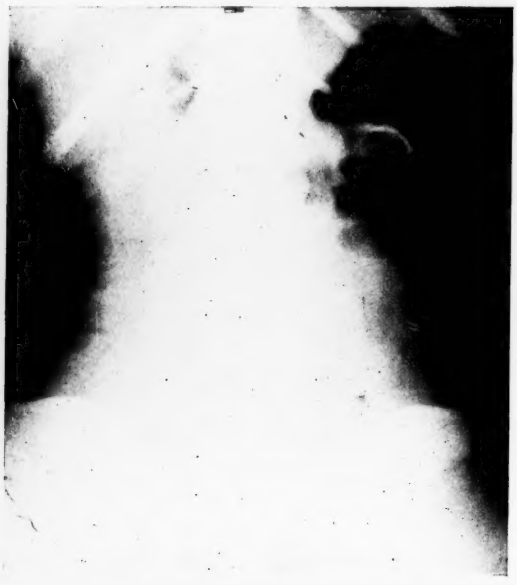

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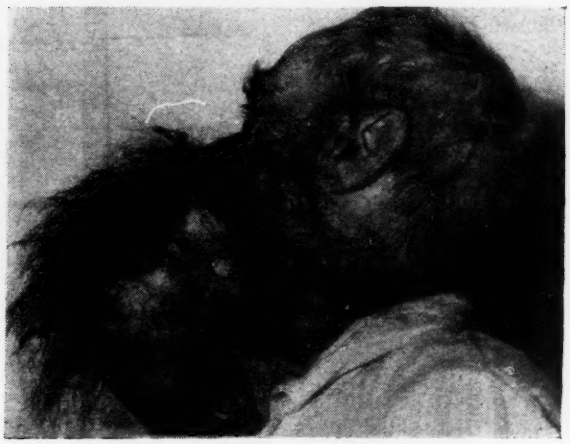

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sac contained the caecum and portion of the transverse colon and ileum. The tumour weighed 11 pounds (5000 grammes). An incision was first made on the infero-medial aspect of the tumour, and the hernial sac and its neck, and the femoral vessels and nerve were defined. The sac was opened, and its contents were returned to the peritoneal cavity. The sac was closed after redundant tissue had been excised. The deficiency in the abdominal musculature was closed by a piece of tantalum mesh six by four inches in size; this was sutured in place by stainless steel wire, an arcade being left for the femoral vessels. The vascular pedicle of the tumour was then identified, clamped and divided, and the remainder of the tumour was dissected out and removed. The large defect in the skin after removal of the tumour was closed by bringing the skin edges together in a star-shaped fashion.

The patient has since remained well and active (Figure III).

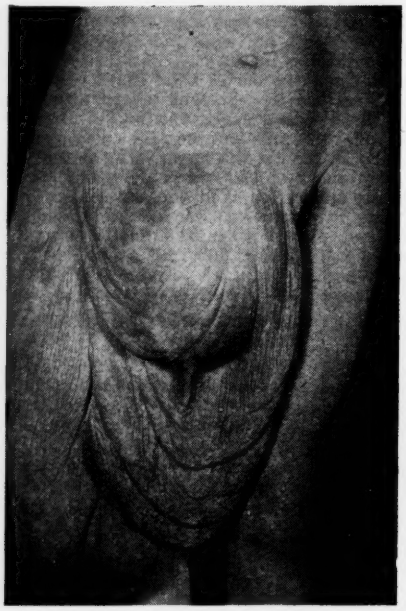


FIGURE IA.
Anterior view of large neurofibroma of right groin, with traction hernia.

FIGURE IB.
Lateral view.

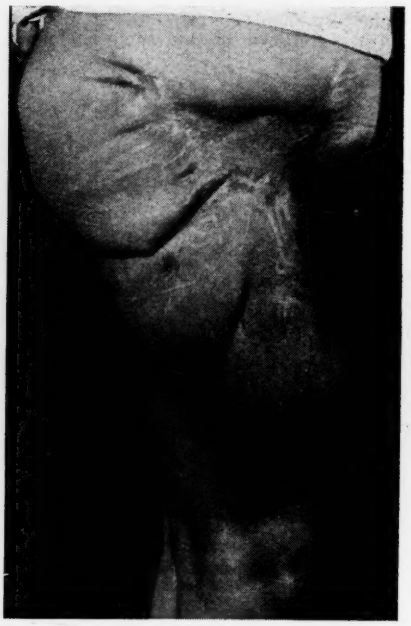


FIGURE III.
Post-operative result.

The pathological report was as follows.

A large soft tumour covered with wrinkled skin was received. Most of the tumour consisted of soft pink fibrous tissue which extended to the cut surface over a large area. There was one hard region with evidence of past hæmorrhage.

Microscopic examination: The lesion is a variety of neurofibroma very suggestive of neurofibromatosis. It consists of loose, œdematous, well-differentiated nerve sheath tissue intermingled with adipose and fibrous tissue. In the neighbourhood of the region of organizing hæmorrhage the tissue is cellular, but I think this is to be interpreted as a reaction to injury and not as evidence of activity in the tumour, which elsewhere appears benign.

Acknowledgement.

I am indebted to Dr. E. Cortis and to the resident medical staff and the nursing staff of the Sydney Hospital for their assistance, and to Mr. R. Money for the photographs.

Legend to Illustration.

FIGURE II.—Skiagram after barium meal, revealing the intestinal contents of the traction hernia.

JEJUNAL VOLVULUS OCCURRING IN BOWEL WITH A NORMAL MESENTERY.

By THOMAS F. ROSE.

From the Department of Surgery, the Royal North Shore Hospital of Sydney, Sydney.

IN a recent series of 39 cases of small bowel volvulus with a normal mesentery, in 33 the ileum was involved, in one all the small bowel and in five the jejunum (Rose, 1955). Jejunal volvulus is thus a rarity, and in ten years only two instances occurred at the Royal North Shore Hospital of Sydney. However, in the next three months, three more were encountered. It is the purpose of this paper to discuss these five cases of jejunal volvulus.

Causation.

Predisposing causes were present in all five instances. In three (Cases I to III), the volvulus was due to a single, short, tough, band-like adhesion, probably of developmental origin, uniting the apex of the involved loop of bowel to the posterior abdominal wall in Cases I and II and to the anterior abdominal wall in Case III. In Cases I and II the patients had not had any prior abdominal operation or any abdominal illness. The third patient (Case III) had had an appendicectomy thirteen days previously for a ruptured gangrenous appendix with peritonitis localized to the right iliac fossa and pelvis. The adhesions due to this were friable and thin and obviously recent, whereas the jejunal band was tough and fibrous and obviously old, and well away from the recently involved area of the abdomen. In two instances (Cases IV and V) the volvulus was due to post-operative inflammatory adhesions, in Case IV following an appendicectomy for acute appendicitis six years previously, and in Case V occurring six weeks after a Wertheim hysterectomy complicated by a pelvic abscess.

Known exciting causes were present in three instances only. In two (Cases III and V), prior to the occurrence of the volvulus, there was prolonged distension of all the small bowel due to recent adhesions. In Case V, this was accentuated by the spontaneous closure of an ileal fistula. In the third (Case IV), the removal of a large uterine fibroid tumour caused an alteration in the position of the bowel with its adhesions.

Age and Sex.

There were three female and two male patients, all aged between thirty and fifty-four years.

Symptomatology.

In all these cases, the attack for which operation was performed was the first and only one. Owing to the high position of the volvulus, the symptoms were severe from the commencement, and rapidly became worse until operation relieved them.

The primary symptom was pain, which was severe from the start. Commencing intermittently, it became more constant as the mesenteric twist tightened. The position of the pain varied from patient to patient. In two (Cases III and V), the pain was generalized all over the abdomen; in two (Cases I and II), it commenced in the epigastrium and radiated to the left hypochondrium and then to the left iliac fossa; in one (Case IV), it was epigastric only. In no instance was there any pain in the back.

Shock was severe in three patients (Cases III, IV and V), and in Case III it actually lasted for four days after operation in spite of treatment. It was due to three factors—firstly to impulses from the twisted mesentery, secondly to loss of protein, blood cells and fluid into the abdominal cavity as a blood-stained transudate, and lastly to the vomiting due to the high intestinal obstruction causing much fluid and electrolyte loss. In one instance (Case IV), so much blood-stained fluid was lost that the clinical picture for a time resembled that of an intraperitoneal hæmorrhage.

Copious vomiting due to the high intestinal obstruction was a characteristic of all cases, and became worse as the illness progressed.

Tenderness was present as an early sign, and was mostly high up on the left side of the abdomen. However, in Case IV, in which a long volvulus stretched across to the right, the tenderness was in the right hypochondrium.

There was no rigidity in any case, as operation was performed before peritonitis could ensue in each instance.

In two patients (Cases I and II), the distension was present in the upper part of the abdomen, especially on the left side. In one (Case IV) there was a large, tender, cystic mass in the right hypochondrium, this position being later shown to be due to the long loop of involved jejunum crossing the mid-line. In two patients (Cases III and V) there was generalized abdominal distension due to dilated small bowel present before the onset of the volvulus. In Case III, this distension increased on the left side of the abdomen with the onset of the volvulus.

In Case I, on radiographic examination, fluid levels actually in the volvulus were seen just below the left costal margin; in Case IV, the levels in the volvulus were seen to be just below the right costal margin because of the long loop of bowel forming the volvulus, whereas dilated jejunum above the volvulus was seen in the left hypochondrium (see Figure I). In Cases III and V, generalized fluid levels were present before the volvulus occurred, the small bowel being already obstructed.

Duration of History.

The length of time between the onset of the illness and operation varied from four to twenty-four hours. In four instances the bowel was perfectly viable; but in Case IV, in which the period was twelve hours, though the bowel appeared viable, a small perforation occurred on the sixth post-operative day, causing peritonitis and death. It is therefore obvious that in the other four cases the twist was not a tight one, as otherwise the bowel would not have been viable.

Diagnosis.

In Case IV the diagnosis was first thought to be an intraperitoneal hæmorrhage; but the correct diagnosis was made as soon as the cystic mass of volvulus was palpated shortly after the patient's admission to hospital. In all the other cases the correct diagnosis of a high small bowel strangulation-obstruction was made, but the cause was not realized until operation was performed.

Treatment.

Routine stomach suction and parenteral therapy as a preoperative measure for intestinal obstruction was instituted immediately on the patient's admission to hospital. This was reinforced by plasma or dextraven given intravenously when shock was present. Whole blood was given in Case IV, in which the picture of an intraabdominal hæmorrhage first presented. As soon as fluid and electrolyte balance was improved, abdominal exploration was performed. Gastric suction and parenteral therapy were continued after operation until peristalsis returned.

At operation copious blood-stained free peritoneal fluid was found, which in Case IV looked like pure blood. In each instance a jejunal volvulus was found in the upper left quadrant of the abdomen. The length varied from one to three feet, the latter length being found in Case IV, in which the apex of the volvulus lay in the right hypochondrium. In Cases I and II the twists were in an anticlockwise direction, whereas in the other three the twists were clockwise.

The volvulus in each instance had the usual appearance of viable strangulated bowel, and normal circulation rapidly returned as soon as it was untwisted, even in Case IV; however, in this case six days later an unnoticeable tiny necrotic area perforated, causing peritonitis.

The apex of the volvulus was attached to the posterior abdominal wall by an old developmental adhesion in Cases I and II, and to the anterior abdominal wall in Case III. In Case IV there were numerous generalized old inflam-

matory adhesions between the coils of bowel, and under one, attached by both ends to the posterior abdominal wall, a loop of jejunum had slipped and then twisted. In Case V there were numerous recent inflammatory adhesions and a pelvic abscess. The apex of the volvulus was attached to the previous abdominal incision by one of these adhesions.

In Cases III and V, generalized small-bowel distension was already present, owing to recent inflammatory adhesions situated mostly round the ileal loops.

In each instance, the responsible adhesion was divided and the volvulus untwisted. In addition, the adhesions causing the prior obstruction in Cases III and V were divided, and a pelvic abscess in Case V was drained.

Mortality.

Two patients died. In one case (IV), bowel thought to be viable actually perforated through a tiny hole six days after operation, and the patient rapidly died of general peritonitis. In common with many cases of post-operative peritonitis, there were few signs to implicate the peritoneal cavity—only a rapid pulse rate and signs of shock, which responded temporarily to nor-adrenaline. In the other case (V), death was due to the previously existing sepsis and ileal fistula, and not to the volvulus, which at autopsy was found not to have recurred.

Autopsy in both these cases revealed a perfectly normal mesentery.

Follow-up Examination.

One patient has been followed up for three years, one for two years, and one for seven months, and all have been well, with no evidence of recurrence of the volvulus.

Clinical Records.

Cases I and II have already been reported fully in this journal (Rose, 1954), so that only a summary of each need be given here. Neither patient had had any previous abdominal operation or illness. The predisposing cause in each case was a single, tough band, probably of developmental origin, attaching the apex of the volvulus to the posterior abdominal wall on the left side. In neither instance was there an apparent exciting cause.

CASE I.—In Case I the patient was a married woman, aged thirty years, who had a twelve-hour history of severe, recurrent abdominal colic commencing in the epigastrium, and radiating to the left side of the abdomen. These attacks of pain were becoming more frequent and severe, so that they were almost continuous. Each attack was accompanied by copious vomiting and audible borborygmi. Examination of the abdomen disclosed distension of the upper part of the abdomen, most pronounced on the left side, where tenderness was elicited. A plain radiograph of the abdomen showed three fluid levels below the left costal margin. Dilated small bowel was seen to occupy the left side of the abdomen.

At operation, a volvulus of a loop of the upper part of the jejunum was found, twisted through 360° in an anti-clockwise direction. A single, thick fibrous band bound the ante-mesenteric border of the apex of the loop to the posterior abdominal wall on the left side. As the bowel was viable, all that was required was to divide the adhesion and untwist the volvulus. Convalescence was uneventful, and a follow-up examination of the patient two years later revealed no recurrence of symptoms.

CASE II.—The clinical history in this case was similar to that in Case I, except that it extended over twenty-four hours. The abdominal distension and tenderness were more widespread, involving all the left side of the abdomen. No radiographs were taken. The operative findings and treatment were essentially the same. Convalescence was uneventful, and a follow-up examination three years later revealed no recurrence of symptoms.

In Case III the patient had had a prior abdominal operation for the removal of a ruptured gangrenous appendix through a McBurney incision thirteen days previously. The predisposing cause was a single, tough band, probably developmental in origin, attaching the apex of the volvulus to the anterior wall in the left upper quadrant. The exciting cause was prolonged small-bowel distension due to recent adhesions in the right iliac fossa from the gangrenous appendix and its removal.

CASE III.—This male patient, aged thirty-three years, had a four-day-old ruptured gangrenous appendix with pelvic peritonitis. Appendicectomy with drainage was performed.

During the next thirteen days there were recurrent episodes of partial lower small-bowel obstruction characterized by mild colic, vomiting and generalized small-bowel distension, which was confirmed by radiological examination of the abdomen. These attacks gradually subsided by conservative measures (stomach suction and intravenous therapy) until the thirteenth day, when, after an attack which did not subside, the patient developed sudden, acute generalized abdominal pain, with severe vomiting and shock. The abdominal distension increased greatly, especially on the left side.

Operation four hours later disclosed partial obstruction of the small bowel at the terminal portion of the ileum, due to recent fibrinous adhesions. In addition, there was a viable volvulus of the upper part of the jejunum in the left hypochondrium. Its apex was attached to the anterior abdominal wall there by a single tough band. This was divided and the bowel untwisted. About a foot of bowel was involved in the volvulus, which was clockwise. The distal adhesions were separated also. Convalescence was complicated by a severe shock-like state lasting four days, from which the patient gradually recovered. A follow-up examination seven months later showed that the patient was very well.

In Case IV the patient had had two prior abdominal operations—an appendicectomy six years previously for acute inflammation of an unruptured appendix localized to the right iliac fossa, and a hysterectomy sixteen days previously through a lower mid-line incision. The predisposing cause of the volvulus was the presence of generalized old abdominal adhesions, presumably from the appendicectomy. The exciting cause was the alteration in the position of the small bowel with its adhesions following the removal of a very large fibroid uterus.

CASE IV.—A female patient, aged thirty-two years, had been well except for acute appendicitis (without rupture) six years previously, for which appendicectomy through a McBurney incision had been performed. Convalescence after this was uneventful. Sixteen days prior to her admission to hospital, she had undergone a subtotal hysterectomy for uterine fibromyomata, including one pedunculated fibromyoma whose fundus reached well above the umbilicus. Convalescence from this operation was uneventful, the bowels being opened well without purgatives on the fourth post-operative day and daily thereafter. The patient went home on the fourteenth day with her wound well healed.

Two days later, on the day of her admission to hospital, she was seized with sudden, severe colicky epigastric pain, with copious vomiting and collapse. The pain, at first colicky, became continuous and very severe. Three hours after the pain commenced, she was found to be white and pulseless in a state of severe shock. Her abdomen was generally tender, but there was no rigidity or mass present. Her doctor thought that she had an internal hæmorrhage because of the signs of blood loss in the abdomen (later shown to be due to the great loss of blood-stained transudate).

Seven hours later, when she was examined at hospital, the picture of blood loss was paramount. Her pulse rate was weak and the rate was rapid; her blood pressure was 50 millimetres of mercury, systolic, and the diastolic pressure could not be estimated. She was complaining of severe, continuous upper abdominal pain and vomiting. The upper part of the abdomen was distended. There was a rounded, tender, immovable mass in the epigastrium and right hypochondrium. Free fluid was present in the flanks. X-ray films showed two loops of dilated jejunum in the left hypochondrium and two fluid levels in the right hypo-

chondrium which were later shown to be in the volvulus (Figure I).

Operation could not be performed for a further two hours (twelve hours after the commencement of her illness), until her condition was improved by the giving of blood. There was then found an upper jejunal volvulus involving three feet of bowel, which was cyanosed and hæmorrhagic with much free sanguineous fluid. The mesentery was also hæmorrhagic. The volvulus twisted 360° clockwise. Many old adhesions were found among the coils of small intestine; but the cause of the volvulus was a thick adhesion attached at both ends to the posterior abdominal wall like a bucket-handle. The affected loop of bowel had slipped through this adhesion and then twisted. The adhesion was divided and the volvulus untwisted. After some time, the bowel, at first of doubtful viability, appeared viable and was returned to the abdominal cavity. (This was done thankfully, as the patient's condition did not appear to warrant an extensive resection.)

Convalescence was first complicated by paralytic ileus resembling acute dilatation of the stomach, with a rapid pulse rate for the first four days. This was treated in the usual manner by stomach aspiration and intravenous therapy. The patient then improved, and commenced to pass flatus and a little fæces *per rectum*. However, on the sixth day, though with no complaint of abdominal pain, she went into a state of severe shock, and, after the abdomen had become slightly distended, died some hours later.

Autopsy disclosed, surprisingly, that general peritonitis was present from a very small hole on the mesenteric border of the affected jejunal loop. The rest of the loop was viable and the mesentery was normal.

The patient in Case V had had a prior abdominal operation, in that she had six weeks previously had an application of radium to her cervix followed by a Wertheim hysterectomy for a carcinoma of the cervix. The predisposing cause was the presence of post-operative adhesions, in particular one running from the apex of the volvulus to the abdominal scar. The exciting cause was small-bowel distension due to a pelvic abscess and adhesions accentuated by spontaneous closure of an ileal fistula.

CASE V.—The patient, a married woman, aged fifty-four years, had had a carcinoma of the cervix treated by radium application followed by a Wertheim hysterectomy. This was complicated by intraabdominal sepsis with small-bowel distension, culminating in a pelvic abscess and an ileal fistula. Six weeks after operation the fistula closed spontaneously, which led to an increase in the abdominal distension. Two days later the patient developed sudden severe generalized abdominal pain, with vomiting and pronounced shock. Examination of the patient disclosed generalized abdominal distension and also tenderness. A rectal and pelvic examination disclosed a tender mass in the pouch of Douglas. Radiographic examination disclosed distended loops of small bowel with fluid levels all over the abdomen.

At operation, eighteen hours after the commencement of the pain, a large pelvic abscess was found, with generalized distension of the small bowel. The abdomen was full of recent adhesions. In the jejunum was a viable volvulus, one foot long, twisted 180° in a clockwise direction. From its apex was a firm adhesion running to the previous abdominal incision. The adhesion was divided, the volvulus untwisted and the abscess drained.

The patient died two days later from the preexisting sepsis. Autopsy disclosed that the volvulus had not recurred and that the mesentery was normal.

Acknowledgement.

I wish to thank Dr. Noel Fowler, F.R.C.S., for permission to quote the details of Case III; this patient was under his care at the Royal North Shore Hospital of Sydney.

Reference.

ROSE, T. (1954), "Volvulus of the Jejunum Occurring in Bowel with a Normal Mesentery: A Report of Two Cases", M. J. AUSTRALIA, 1: 557.

ROSE, T. (1955), "Volvulus of the Small Intestine with a Normal Mesentery", *Australian & New Zealand J. Surg.*, 25: 41.

Legend to Illustration.

FIGURE I.—Case IV: Radiograph of the abdomen taken with the patient in the erect position. This shows fluid levels on the right side of the abdomen (later shown to be in the actual volvulus, which had crossed the mid-line), together with dilated coils of the jejunum in the upper left abdominal quadrant.

Reviews.

Blutgerinnungsfaktoren. By Erwin Deutsch; 1955. Vienna: Franz Deuticke. 9½" x 6½", pp. 312, with many illustrations. Price: DM42.

THE valuable collection of separate studies in the field of biochemistry published in Vienna was suspended in the thirties, but has been resumed in Volume I of a new series and this is devoted to blood coagulation factors. The subject of blood coagulation is one of those topics to which the clinical contribution is much smaller than that from the biochemical laboratory. The author of this book, Erwin Deutsch, has taken a major part in the complicated investigations which have engaged the attention of researchers in many countries, and the literature of which is not easy to read on account of the wide diversity of terminology employed. Not so long ago we were content with a simple scheme of fibrin ferment or thrombin acting on an ingredient of the plasma called fibrinogen; in the making of thrombin calcium ions were for some reason necessary. But now there have been revealed precursors, active and inactive, cofactors, active and inactive, and accelerators, active and inactive. Erwin Deutsch's main studies have been directed towards Factor VII or convertin (which Alexander named "serum prothrombin conversion accelerator") and its precursor proconvertin. There was a time when botanists who knew no chemistry and chemists who knew no botany thought that by mere exposure of a chlorophyll solution to sunlight a synthesis of carbohydrate from carbon dioxide and water could take place; now it is known that photosynthesis is a most complicated chain reaction. Similarly blood coagulation is the outcome of a lengthy and involved series of interactions. One pities the unfortunate medical student if he is expected to make himself familiar with this formidable entanglement of hypotheses, and one can only hope that some simplification such as Copernicus introduced into solar astronomy and Dalton into chemistry will operate in this field of blood coagulation. The chapter on heparin can be warmly commended. One useful feature is a list of synonyms, for with the 25 factors discussed only one who has mastered the immense output of research in the last dozen years could draw up this valuable table.

Ciba Foundation Colloquia on Ageing. Volume I: General Aspects. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S., assisted by Joan Etherington; 1955. London: J. and A. Churchill, Limited. 8" x 5½", pp. 270, with 38 illustrations. Price: 30s.

THE Ciba Foundation has commenced a new series of colloquia, this time on aging. Volume I has recently been published, and it maintains the high standards of the previous Ciba publications. The volume just published deals with certain general aspects of aging. Thirty-four experts on various aspects of the subject from Europe and America met in London in July, 1954, and papers were given and discussions held. There were sixteen papers read on such subjects as the definition and measurement of senescence, the pathological basis of aging and mental aspects, the effects of aging on various systems such as the respiratory system, the skin and the skeletal system and calcification. Some more general papers dealt with tissue transplantation in relation to aging of the organs of reproduction, nutrition in gerontology, and psychological aspects of aging.

The papers are of the usual high standard, but at a very readable and, for the non-expert, understandable level. The discussions form perhaps the most important parts of the book, particularly as differences of opinion were common and very clearly expressed. The chairman, R. E. Tunbridge, in his opening remarks asks: "Is ageing a chronological term, merely reflecting the passage of years, or are the public right in assuming, as they generally do, that ageing

is synonymous with senescence and/or decay?" In the final summing up he says: "I think we shall have to accept ageing as a chronological process." While the book does not answer such and other questions adequately, it gives particularly interesting reading at least to "those, who, growing old, pontificate upon growing old" as one of the speakers put it.

The Suprarenal Cortex: Proceedings of the Fifth Symposium of the Colston Research Society held in the University of Bristol, April 1st-4th, 1952. Edited by J. M. Yoffey; 1953. London and Sydney: Butterworths Scientific Publications. 10" × 7", pp. 240, with many illustrations. Price: £2 18s. 6d.

SEVERAL books have appeared recently on the adrenal cortex and the steroids derived from it. One of the most recent is the Ciba Foundation Colloquia on "The Human Adrenal Cortex" reviewed in these columns on August 27, 1955. This gave highly technical papers and discussions by experts to experts. The proceedings of another symposium on the same subject have just appeared, although the symposium was held in 1952. This was under the auspices of the Colston Research Society of the University of Bristol. Twenty papers were given by experts in a wide range of subjects connected with the adrenal cortex and the audience was a large one, not wholly composed of experts. The subjects treated included the preparation and properties of ACTH, the nature of adrenal cortical secretion, the relation of the adrenal cortex to the genital organs and mammary gland, various aspects of the relation of the adrenal cortex and its steroids to infection, personality disorders, shock, skin grafting, mineral and water metabolism and homœostasis. The surgery of the adrenal glands is also dealt with as well as the use of corticosteroids and corticotropin in the treatment of rheumatic and other diseases.

While being thoroughly scientific, the treatment is, for the most part, such as to make the communications interesting and understandable to the non-expert. The editor states that "no attempt has been made to conform to the stricter editorial canons". It is a pity that some editing was not done, for it is disconcerting to find the same steroid with two quite different names in different parts of the book with no clue as to their identity. This book can be recommended as quite the best account available of the functions of the human adrenal cortex for the general medical reader.

Midwifery. By Ten Teachers, under the direction of Frederick W. Roques, M.D., M.Chir., F.R.C.S., F.R.C.O.G., edited by Frederick W. Roques, John Beattie and Joseph Wrigley; Ninth Edition; 1955. London: Edward Arnold (Publishers), Limited. 8¼" × 5¼", pp. 616, with 249 illustrations. Price: 32s. 6d.

THE increasing content of knowledge concerning the science and art of obstetrics has emphasized the need for conciseness and clear thinking in the preparation of an obstetrical text-book suitable for medical students. That this has been achieved by good teamwork and editorial direction is evident in the ninth edition (1955) of "Midwifery" by "Ten Teachers" under the direction of F. W. Roques. Whilst the preface to this edition does not make the usual claim that it has been thoroughly revised and rewritten, reference to the text makes it clear that this has indeed been done.

A few examples may be given; thus reference is made to the value of the discreet use of œstrogens in the control of breast engorgement, exchange transfusion for erythroblastosis is dealt with in detail, the scope and limitations of antibiotic therapy are discussed with proper regard to "drug resistance", the section on disordered uterine action is written in an enlightened fashion, placental insufficiency is recognized and "obstetrical anuria" is adequately dealt with. The avoidance of Cæsarean section for the delivery of patients whose pregnancy is complicated by heart disease is rightly stressed, and the management of the pregnant diabetic woman is discussed in the light of modern principles. Such examples could be multiplied indefinitely—they merely serve to indicate the manner in which the subject is dealt with.

Because a student's text-book must necessarily be written in a dogmatic fashion, criticism in matters of detail should be limited and the general method of presentation only subjected to close scrutiny. There is, however, only one criticism on the latter score: the three chapters dealing with mild and severe preeclampsia, eclampsia, essential hypertension and chronic nephritis are all dealt with under the heading "Albuminuria in Pregnancy". At first sight this emphasis on proteinuria would appear to stress unduly this aspect of the diagnosis of the conditions referred to, and this at a time when most teachers are stressing that albuminuria is a late sign of pregnancy toxæmia. The actual content of the three chapters, however, is written in an enlightened and helpful manner that deserves a better heading.

With the more liberal use of Cæsarean section for *placenta prævia* and for non-recurring indications in primiparæ, one would like to see a fuller discussion on the conduct of vaginal delivery after previous lower segment Cæsarean section. It is pleasing to see the Lovsett technique for the management of extended arms fully described, and a short section on excessive involution of the uterus after childbirth and lactation brings to light an interesting and not entirely unusual condition. The book with its modern concise presentation of the subject is probably one of the best students' text-books of obstetrics available at present.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Diagnosis of Congenital Heart Disease: A Clinical and Technical Study by the Cardiologic Team of the Pediatric Clinic Karolinska Sjukhuset, Stockholm", by Sven R. Kjellberg, Edgar Mannheimer, Ulf Rudhe and Bengt Jonsson; 1955. Chicago: The Year Book Publishers, Incorporated. 10" × 7", pp. 674, with 581 illustrations. Price: $22.00.

Based on the study of 396 cases of congenital heart disease.

"Advances in Internal Medicine", edited by William Dock, M.D., and I. Snapper, M.D.; Volume VII; 1955. Chicago: The Year Book Publishers, Incorporated. 9" × 6", pp. 312, with many illustrations. Price: $8.50.

The subjects dealt with are eight in number, each subject is discussed under sectional headings and many references are given in full.

"Roentgen Interpretation", by George W. Holmes, M.D., and Laurence L. Robbins, M.D.; Eighth Edition; 1955. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6", pp. 526, with 371 illustrations. Price: £5 10s.

The first edition of this book was published in 1919.

"Biochemistry and the Central Nervous System", by Henry McIlwain, Ph.D., D.Sc.; 1955. London: J. and A. Churchill, Limited. 9½" × 6", pp. 280, with 43 illustrations. Price: 40s.

The subject of the book forms part of teaching in physiology and in psychological medicine in the University of London.

"Ciba Foundation Symposium on Experimental Tuberculosis Bacillus and Host, with an Addendum on Leprosy", editors G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S., assisted by Cecilia M. O'Connor, B.Sc.; 1955. London: J. and A. Churchill, Limited. 8" × 5¼", pp. 408, with many illustrations. Price: 42s.

Twenty-two subjects were discussed at the symposium; four are included in the addendum.

"Breast Cancer and its Diagnosis and Treatment", by Edward F. Lewison, B.S., M.D., F.A.C.S.; 1955. London: Baillière, Tindall and Cox. 10" × 7", pp. 490, with 181 illustrations. Price: 114s.

The author's purpose has been to sift available information and combine it with practical experience for all interested in the total care of the disease. There are eight contributors.

"Collected Papers on Aviation Medicine", presented at Aeromedical Panel Meetings of the Advisory Group for Aeronautical Research and Development, Palais de Chaillot, Paris; 1955. London: Butterworths Scientific Publications. 10" × 6", pp. 218, with many illustrations.

The object of the presentation of some of these papers at a meeting of the AGARD Flight Test and Aeromedical Panels was to acquaint aeronautical engineers and research scientists with human limitations which need consideration in aircraft design.

The Medical Journal of Australia

SATURDAY, DECEMBER 10, 1955.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

PETHIDINE.

THOSE who have followed the discussions of the Federal Council of the British Medical Association in Australia will know that the use of pethidine has been causing much concern in public health circles, as well as among the councils of the Association. Concern has also been felt in other countries and this is reflected in the pronouncements of the World Health Organization. At the last meeting of the Federal Council pethidine was discussed at some length as the result of a request from the Director-General of Health, Canberra, that consideration should be given to the sponsoring of a special committee which should ascertain the reasons for the increase in the prescribing of pethidine. There has actually been a phenomenal rise in the consumption of pethidine. In 1949 and 1950 the consumption per head of population in Australia was less than that of the United Kingdom, the United States of America, New Zealand and Canada. In a recent survey it was found, however, that the average consumption per head of population in the United Kingdom, the United States of America, New Zealand and Canada was only 61% of the consumption per head of population in Australia. This means that from 1951 onwards there has been a considerable increase in consumption in Australia, and in 1953 Australia had the unenviable distinction of being the highest consumer per head of population.

Pethidine, which is the ethyl ester of 1-methyl-4-phenylpiperidine-4-carboxylic acid, was introduced by Eisteb and Schaumann in 1939. It is administered as the hydrochloride. Martindale's "Extra Pharmacopœia" gives as synonyms meperidine hydrochloride, dolantin and isonipecaine; proprietary names are "Dolantel" and "Demerol". "The Extra Pharmacopœia" discusses it together with amidone and phenoxadone. In general its pharmacological actions on the pupil, heart, bronchi and vagus nerve resemble those of atropine, while its effects on bronchi, intestine and blood vessels are similar to the spasmolytic effects of papaverine. It resembles morphine in its analgesic, sedative and euphoric properties. Pethidine is, perhaps unfortunately, relatively non-toxic, though it may give rise to dizziness, sweating and dry mouth; nausea and vomiting occur, but rarely. Intravenous injection generally causes vasodilatation and a fall in blood pressure. Addiction to pethidine is rapidly acquired and in severe cases is as hard to break as morphine addiction. In "The Extra Pharmacopœia" reference is made to a report by P. Polonio, published in *The Lancet* in 1947 (Volume I, page 592) on 15 cases of pethidine addiction. The treatment given included immediate withdrawal of the drug, modified insulin treatment and appropriate symptomatic treatment for the disturbing side-effects. Polonio stated that relapses were common and that psychotherapy and occupational therapy were necessary. J. Solis is also recorded as having reported a case in which a woman of forty-eight years became an addict. In her case withdrawal symptoms were severe, the psychological symptoms appearing less intense and the physical symptoms more intense than with morphine. In these circumstances it is not to be wondered at that pethidine has been classified in the same category as morphine and diacetylmorphine under the International Narcotic Drug Conventions and that it is subjected to the same regime as morphine in the *Dangerous Drug Acts* of the several States of the Commonwealth. It is important to note that with the great increase in the consumption of pethidine in the last five years there has been no proportionate reduction in the consumption of morphine. Thus in the year 1949 the amount of pethidine consumed was 11·65 kilograms per million of population and the amount of morphine 16·30 kilograms; in the years 1953-1954 the amounts were respectively 42·75 and 14·70 per million of population.

At its last meeting the Federal Council agreed to the suggestion of the Director-General of Health about the appointment of a special committee. The committee consists of Dr. Byron Stanton (of Melbourne), Dr. A. W. Morrow and Dr. A. J. Murray (both of Sydney) and Dr. C. E. Cook (of the Commonwealth Department of Health). It has, we understand, met, and its decisions will be awaited with the greatest interest. The wishes of the members of the Federal Council expressed in the discussion have been met—the majority of the members of the committee are members of the practising profession, "top line" men have been chosen and their selection was not entirely in departmental hands. But the profession does not need to wait for a report from this committee before it decides what has to be done. It is true that pethidine is used in obstetrics, as members of the Federal Council pointed out, but this would not account for the enormous increase in the amount used during recent years. Possibly a certain amount of pethidine has been used in place of heroin which, during the years referred to, was being put on one side, but the amount would not be large.

It is thought likely that some practitioners may still think that pethidine is less likely to lead to addiction than morphine. This view was expressed in a report by the World Health Organization. The extract from the report is as follows:

> The Expert Committee on Drugs Liable to Produce Addiction having considered a report on pethidine addiction as encountered at the Public Health Service Hospital, Lexington, Kentucky, U.S.A., and noting the high incidence of such addiction among members of the medical, nursing and associated professions, considered that an important factor in the development of pethidine addiction not only in the United States of America but also in other countries has been the attitude of physicians towards the drug, based upon the widespread belief that it is less dangerous in this respect than morphine.

The report also made recommendations to all governments and to the profession throughout the world:

> Being convinced that experience with the drug, both in experiments and in clinical practice, is contrary to this belief, the Expert Committee was of the opinion that pethidine is as dangerous as morphine as a potenital addicting agent, that its use should be undertaken only with full realization of this danger, and that its administration should be approached with the same attitude and attended by the same precautions as are recognized for morphine.

A point which may be of importance in this country is that pethidine is one of the drugs available under the provisions of the *Pharmaceutical Benefits Act*. It is possible that more tablets than are needed may be ordered for a patient and also that, because of its ready availability, pethidine may be prescribed when some other drug, potentially less dangerous, would meet the case. Pethidine cannot be obtained by patients without a doctor's prescription, and ultimately therefore the responsibility for its over-use must rest with the medical practitioner. The subject need not be laboured any further. One thing is quite clear—that if the use of pethidine increases still further, steps will have to be taken by the authorities to make it more difficult to obtain supplies, for example, to forbid the manufacture of tablets and to issue only ampoules of solution. The matter rests with the profession.

Current Comment.

ACCIDENTS IN CHILDHOOD.

READERS of this journal will recall the series of articles on accidents to pre-school children by F. W. Clements, of the Institute of Child Health, Sydney, published in this journal in March of this year. They will be interested in an article by H. A. Carithers[1] on accident prevention in childhood. Carithers bases his remarks on insurance company statistics and he states that whereas in 1930-1931 pneumonia and influenza caused 126·4 deaths per 100,000 in childhood, these diseases caused only 15·3 deaths per 100,000 in 1951-1952. On the other hand, accidents third on the list at the earlier period with 55·8 deaths per 100,000 topped the list in 1951-1952 with 28·9. Carithers points out that the reduction of 48% in deaths from accidents is not an index of fewer accidents, but of better treatment's saving more lives, and he compares this figure unfavourably with the reduction of 88% in pneumonia and influenza, and 93% in gastro-enteritis. Carithers also states that for every fatal accident among children there are at least 100 non-fatal ones, of which four result in some permanent disability.

An analysis of accidental deaths in the one-to-four years age group shows the causes to be: motor vehicles, 37·2%; burns, scalds and conflagrations, 19·9%; drowning, 15·4%; falls, 6·0%; poisoning, 5·2%; choking, 4·4%; and others, 11·9%. Of these, Carithers concentrates on poisoning, which is due in almost every instance to the child's having swallowed some substance left available to it by carelessness of adults, and is thus truly preventible. Causes of accidental poisoning in children in the United States have been listed as: aspirin, 17·3%; other medications, 25·7%; kerosene, 16·0%; ant and rat poisons, 10·6%; others, 30·4%. The prominence of medications in this list is understandable—many have a pleasant taste (or, at any rate, the child is persuaded that they are pleasant), many resemble lollies, and all have been recommended to children as good things. Ant and rat poisons are frequently put out on scraps of foodstuffs, which the child finds in hidden corners and naturally picks up. The high status of kerosene in the list is harder to understand, but it is commonly used for heating and cooking, and has a very familiar place in many households; Carithers, in a local survey, found that paradoxically kerosene poisoning was commoner in the summer than in the winter. His explanation, obviously a sound one, is that small children drink kerosene (and other household liquids) because they are thirsty, and suggests that the provision of plenty of drinks in hot weather will remove the stimulus to seize on any available fluid (which, nevertheless, should not be allowed to be available, let it be emphasized). This ties up with the cause of many scaldings—children pull saucepans off the stove, or drink from the spouts of kettles and teapots, because they are thirsty.

We are now in a position to summarize the means of preventing the most common home accidents of childhood. First, all medications should be kept locked up, because they taste nice, or look like lollies. Secondly, small children should not be allowed to be thirsty, lest they drink undesirable liquids. Thirdly, no rat or ant poisons should be laid down in households where there are small children, and especially they should not be placed on scraps of foodstuffs. Here we do well to recall the contention of Clements that failure of the adult to put himself or herself in the position of the child is a factor in all accidents of childhood. Clements also made the reasonable statement that protective actions by adults are not enough, but that during the second year of the child's life deliberate education of the child in regard to the hazards of accident should be undertaken.

[1] *J.A.M.A.*, September 10, 1955.

HÆMOGLOBIN AND MUTATIONS.

THAT gene mutations lead to the formation of new proteins in enzymes or special tissue proteins is well recognized, but there are at present few occasions on which one can isolate these new proteins and compare them with the proteins produced by the non-mutated gene. The various kinds of hæmoglobin which have been isolated from cases of hereditary hæmolytic anæmia are examples of such new proteins, and a study of the genetics of their appearance is very useful. L. Pauling in 1946 suggested that the hæmoglobin in the red cells of patients with sickle-cell anæmia was of different constitution from normal adult hæmoglobin, and in 1949 L. Pauling, H. A. Itano, S. J. Singer and I. C. Wells described the separation and properties of this new hæmoglobin. Since then fifteen more hæmoglobins have been described, mostly by Itano and his co-workers. In the Minot Lecture to the American Medical Association in 1955 H. A. Itano describes[1] some of these hæmoglobins, the clinical conditions with which they are associated, and the genetics of their occurrence. The human adult hæmoglobin differs from the form present

[1] *Arch. Int. Med.*, September, 1955.

in the fœtus or newborn in certain physical and chemical properties. The newly found abnormal hæmoglobins associated with anæmias differ from normal adult hæmoglobin in similar ways. The most useful method for the detection of abnormal hæmoglobins has been electrophoresis. Tests for solubility and salting out under fixed conditions are also useful. The hæmoglobins differ from each other in their globin portions, but the exact nature of the differences is not yet known. Hæmoglobin A (normal adult hæmoglobin) is the only form present in measurable amounts in the majority of humans after the first few months of postnatal life. It is also present in some of the abnormal states. Hæmoglobin F (fœtal hæmoglobin) is the first form of hæmoglobin formed in the fœtus. The production of hæmoglobin F persists into the adult states in many cases of hereditary anæmia. It differs from all other human hæmoglobins by its increased resistance to denaturation by alkali and in other ways. Hæmoglobin S (sickle-cell hæmoglobin) differs from A and F in its solubility and electrophoretic behaviour. In the deoxygenated state the solubility of hæmoglobin S is very much less than that of hæmoglobin A. In the deoxygenated red cells it crystallizes out in molecular aggregates, which causes the sickling of the cells. Hæmoglobin C is more soluble than hæmoglobin A. Hæmoglobin D is indistinguishable from hæmoglobin S by electrophoresis, but its solubility is like that of hæmoglobin A. Hæmoglobin E has electrophoretic mobility like that of C on the alkaline side and S on the acid side of their isoelectric points. Hæmoglobin G is much the same as F, but it is labile to alkali. Hæmoglobins H, I and J differ from the others in their electrophoretic mobility and also from each other. Abnormalities in hæmoglobin metabolism may be grouped under three categories: (*a*) the asymptomatic carrier states, (*b*) the sickle disease complex, and (*c*) the thalassæmia and thalassæmia-like complexes. The carrier or trait conditions are due to the presence of a single abnormal gene, and the subjects do not have symptoms referable to the carrier state, but the offspring of the marriage of any two carriers may suffer from chronic anæmia. Sickle-cell trait is shown in about 9% of American Negroes, so the mutation must have occurred a long while ago. The red cells show sickling, but there is no other abnormality. Hæmoglobins A and S are present. Hæmoglobin C trait has been detected in 2% to 3% of American Negroes. Hæmoglobin D trait has been noted in only two families. Hæmoglobin E trait is rare in Europe but common in Thailand and other eastern countries. Hæmoglobin G trait has been found in one person only. Hæmoglobin I trait was found in three generations of an American Negro family, and hæmoglobin J in two generations of another Negro family.

It is to be noted that these abnormal hæmoglobins occur much more commonly among Negroes than in other people. Thalassæmia minor or Cooley's trait is due to another gene mutation. The erythrocytes are microcytic and are less resistant to lysis than normal cells. Sickle-cell anæmia is due to the presence of an abnormal gene producing hæmoglobin S from both parents. Sickle-cell hæmoglobin C disease and sickle-cell hæmoglobin D disease have been observed. In these cases the hæmoglobin S gene comes from one parent and hæmoglobin C or D gene from the other parent. Sickle-cell thalassæmia disease similarly has the two genes from the two parents. Thalassæmia-like conditions occur with one gene for thalassæmia and the other gene for one of the abnormal hæmoglobins other than S.

Two factors then are necessary for a subject to have hereditary hæmolytic anæmia, one abnormal gene from each parent, either the same or different. One of the factors which helps to produce the condition of anæmia is the much slower rate of synthesis of the abnormal hæmoglobins than of normal hæmoglobin in spite of increased stimulus to hæmopoiesis. The mean span of life of the red corpuscles in hæmolytic anæmia is generally much shorter than that of normal cells. The discovery and study of sickle-cell hæmoglobin provided for the first time the explanation for the manifestations of a particular disease on the basis of the physical properties of an abnormal molecule. The synthesis of this abnormal molecule is due to the presence of a mutant gene in man. Further study of these abnormal hæmoglobins and their production may lead to a better understanding of the new proteins, such as enzymes, produced by other gene mutations. They can also give some information as to the spread of mutant genes in the community, if mutant genes are, as is believed by many, produced by atomic radiations as well as other unknown causes.

HORMONES AND THE MANAGEMENT OF PREGNANCY IN DIABETICS.

ONE of the main problems associated with pregnancy in the diabetic woman is the high fœtal mortality. In recent years greatly improved results in this regard have been attributed to hormone therapy. For example, in 1953 Priscilla White, Philip Koshy and Janine Duckers,[1] in a report from the Joslin Clinic in Boston, stated that their fœtal survival rate was 90%—a remarkably high figure, which they associated with their current practice of giving sex endocrine therapy to all pregnant diabetic women except those with borderline diabetes requiring no insulin. It should be noted that this is only one of six rules which have been developed in the Joslin Clinic, "with improved results". The others are: (i) classification of the patient for fœtal and maternal hazard; (ii) treatment of the diabetes to achieve the best possible chemical control; (iii) installation of measures to prevent or correct œdema and hydramnios; (iv) early timing of the delivery; (v) provision of special care for the infant in the immediate post-natal period. These points are mentioned because not everyone is satisfied about the value of the endocrine therapy, despite the convincing theoretical reasons put forward for it. On the other hand no one doubts the outstanding quality of the diabetic management achieved by White and her colleagues at the Joslin Clinic; so that, as F. H. Hales Wilson[2] remarked, in a paper read at a meeting of the New South Wales Branch of the British Medical Association last year, "whether the excellent results are due rather to superb cooperation between patient, obstetrician, physician, pædiatrician and nurses remains to be proved". Wilson went on to refer to a Medical Research Council investigation of the hormone therapy, the results of which had not then been published, and said that he was informed that they did not establish its value. The results have now been published[3] and confirm the information quoted. The investigation, like several recent Medical Research Council investigations, was carried out in a number of centres simultaneously, the results being pooled. Comparable groups of patients suffering from diabetes, but free from major cardio-vascular complications, attending nine hospitals in the United Kingdom were stratified by age and parity and randomly divided into "hormone-treated" and "non-hormone-treated" groups. The former group received tablets containing graduated doses of ethisterone and stilbœstrol; the latter were given inert but otherwise identical tablets. In all other respects the management of the pregnancy was the same. All the patients, 76 in the hormone-treated group and 71 in the control group, were followed for at least six months after delivery. The frequency of stillbirth and neonatal death in the two groups proved to be almost the same, with a total death rate of viable fœtuses of 24% in the hormone-treated group and 26% in the other. Although birth weights were on the average equal, there were four congenital malformations in the treated group and seven among the controls; the frequency of malformation associated with fœtal loss was, however, the same, being three in each group. Diabetic control during pregnancy was equally good in the two series, and the incidence of hydramnios, œdema and albuminuria was equal. There

[1] *M. Clin. North America*, September, 1953.
[2] M. J. AUSTRALIA, July 17, 1954.
[3] *Lancet*, October 22, 1955.

was no significant excess in the incidence of toxæmia in the control group, and the trends in blood pressure readings during pregnancy were identical.

In view of the facts that oral administration of the hormones was used in this trial and that White prefers intramuscular administration, it is important to note that assays were carried out to measure the urinary output of pregnandiol and chorionic gonadotrophin. The report states that although the methods of pregnandiol estimation are not specific, the assay results suggest that either one or the other, or both, of the orally administered preparations was absorbed, but that this had no effect on the pattern of hormone excretion associated with fœtal loss. From these observations it is concluded that oral administration of stilbœstrol and ethisterone, in the doses used (which were determined on the basis that they should have physiological effects similar in nature and intensity to those of the parenteral dosage of stilbœstrol and progesterone used by White), do not reduce fœtal mortality in diabetic patients, and have little, if any, beneficial effect on maternal health in pregnancy.

This is theoretically the main finding of the report, and the point seems to be well established. The disappointing aspect, and perhaps the more important practical finding of the report, is that the proportion of pregnancies ending in live births was practically identical at nearly 70% in each group; the omission of abortions only raises the survival rate among viable fœtuses to 75% in both series. If the 90% survival rate reported by White and her colleagues is reliable (it does not seem to have been questioned) and is not the result of hormone therapy, then their management of the pregnancies must be vastly superior in some other respects. Exactly what those are requires urgent determination. We hope that the Medical Research Council investigators will not let the matter rest at its present negative point.

BIOPSY OF THE KIDNEY.

IT is not only requisite to perfect a new technique, but it is also essential to prove that its performance will be necessary and worth while. R. C. Muehrcke, R. M. Kark and C. L. Pirani[1] have produced a modification of Iversen's method of taking biopsy specimens, percutaneously, from a kidney, which is obviously satisfactory and safe, provided patients are carefully selected. It is in their exposition of the need for such biopsies, and of the benefits to be gained from them, that the authors leave themselves open to some criticism. They first point out the difficulty of making an exact diagnosis of renal disease during life, and the impossibility of diagnosing accurately during life the anatomical changes that will be found in the kidney after death. The first statement would probably be more accurately phrased if the words "pathological change" were substituted for "disease" — what they are in fact concerned with is not the actual disease (which can be diagnosed with satisfying frequency) but the pathological tissue changes involved in the disease process (which are interesting, but rarely essential to diagnosis). Their list of contraindications for kidney biopsy is illuminating in this respect: a bleeding tendency, only one kidney, oliguria with azotæmia, calcific atherosclerosis, perinephric abscess, hydronephrosis, pyonephrosis, renal neoplasm and large cysts. If sufficient investigations have been carried out to cover this list, diagnosis of other conditions could hardly have been missed. Furthermore, of the six case reports which are presented as demonstrating the value of making kidney biopsies, it can be said, without prejudice, that no useful purpose, as regards diagnosis, management, or the patient's welfare, was served. Nevertheless, some information concerning the pathological state of the kidneys was obtained; but it is questionable whether there is anything of value in finding, for instance, that the kidney tissue of a woman with preeclampsia, fully diagnosed clinically, showed changes consistent with preeclampsia; or that when she had recovered, by all clinical criteria, her kidney tissue was normal.

In these columns it has many times been pointed out that laboratory aids are ancillary to clinical examination and cannot, indeed must not, be allowed to replace careful clinical examinations. To go further, to carry out tests which are not entirely without risk to the patient, when a sure diagnosis has already been made, and when the performance of such tests can give the patient no benefit at all, is surely inexcusable. This technique of kidney biopsy, on the evidence submitted, appears to have a value almost entirely limited to academic pathology; its use, not as a routine test in renal disease but as an aid to research, carried out on volunteers who fully understand their position, could perhaps be justified and would probably contribute to our knowledge of renal pathology.

[1] *New England J. Med.*, September 29, 1955.

GAMMA GLOBULIN AND INFECTION.

DEFICIENCY or absence of γ globulin, the plasma protein associated with the antibody activities of the blood, has recently been discussed, in the light of recent work, by David Gitlin.[1] Gitlin classifies these conditions into two groups—primary, in which there is failure in synthesis of γ globulin; in this group there may be congenital agammaglobulinæmia, adult agammaglobulinæmia, and transient or physiological hypogammaglobulinæmia; and secondary, in which there is excessive catabolism or excessive loss from the circulation of γ globulin.

Gitlin discusses fifteen children who had congenital agammaglobulinæmia. All were males; in three instances other males in the immediate family had died, apparently of the same condition, and in three families maternal uncles or male cousins were similarly affected. In one instance three male siblings died of bacterial infections, while the only female escaped. These histories suggest that congenital agammaglobulinæmia is hereditary, recessive and sex linked, with the same inheritance pattern as has hæmophilia. Clinically, the condition is first manifested in infancy or very early childhood by an unusual frequency and repetition of severe bacterial infections, such as pneumonia, septicæmia or meningitis. When the sequence of infections is less severe, the condition may go unrecognized for some time and permanent organic damage may result — for instance, random sampling in a small bronchiectasis clinic revealed two children with agammaglobulinæmia.

Usually γ globulin is not entirely absent in these cases. Ordinary electrophoresis of plasma may fail to reveal any, but more sensitive immunochemical analysis may show that from four to 18 milligrammes *per centum* are present, as compared with the normal level of 600 to 120 milligrammes *per centum*. This minute amount may account for the fact that these children react normally to many virus infections, since, for example, only a very small amount of γ globulin derived from pooled normal plasma, not necessarily convalescent plasma, is necessary for passive protection against measles. An interesting sidelight is that these children lack isohæmagglutinins as well—this fact can be used as a simpler measure for screening for agammaglobulinæmia in those with blood groups O, A and B (not, naturally, with group AB).

That this form of the condition is in fact due to a failure to synthesize γ globulin, and not to excessive loss, was proved first by injecting γ globulin and estimating its loss by catabolism—in these children its destruction was slower than normal; and secondly, by demonstrating the absence of plasma cells, the presumed source of antibody, from the lymph nodes, spleen, and intestinal wall, even after stimulation with specific antigens.

Adult agammaglobulinæmia is similar to the congenital form, but either males or females may be affected, and

[1] *Bull. New York Acad. Med.*, May, 1955.

the range of γ globulin is up to 100 milligrammes *per centum*. It may, of course, be an acquired condition, but Gitlin considers that it may be inherited, becoming manifest only in adult life. Intermediate between this form and congenital agammaglobulinæmia is hypogammaglobulinæmia. Normally, the newborn infant possesses only the γ globulin derived from the maternal circulation; this is gradually metabolized, while the infant does not commence to synthesize its own γ globulin until it is some four to twelve weeks old. If destruction of maternal γ globulin is more rapid than synthesis, or if the commencement of synthesis is delayed, then there may be a period of dangerous hypogammaglobulinæmia, which may be very difficult to detect.

Secondary hypogammaglobulinæmia is best typified by nephrosis, in which there is persistent heavy loss of γ globulin through the renal glomeruli, resulting in a level of only some 200 milligrammes *per centum* in the serum, and of two milligrammes *per centum* in the œdema fluid and 10 milligrammes *per centum* in the ascitic fluid.

Gitlin's survey of this subject is of real value. It is useful to have a systematized classification, and although resistance to infection does not depend on γ globulin alone, or even specifically on the γ fraction of the globulins, since antibodies can also be found in the α and β fractions, and other factors, such as lysozyme, phagocytes and complement enter into the reactions, yet γ globulin does play a very big part. When it is deficient, infections of dangerous magnitude do develop, and intramuscular injections of normal γ globulin, of the order of 100 to 150 milligrammes per kilogram of body weight once a month, confer considerable protection—much more than do antibiotics. On the other hand, γ globulin is of very little value in treatment, whereas here the antibiotics are truly life-saving. But perhaps the most useful aspect of Gitlin's survey is its focusing attention on the necessity for detecting congenital agammaglobulinæmia before permanent damage, or even death, supervenes on the infections which will inevitably occur because of it. Perhaps, also, in infantile hypogammaglobulinæmia, as he defines it, may be the explanation of the rapidity and severity of the infections which sometimes attack premature infants—information on γ globulin levels in such infants may show that injections of γ globulin could be useful in tiding them over a wider gap than usual between the loss of maternal γ globulins and the commencement of their own synthesization.

RECENT THOUGHTS ON THE CHEMOTHERAPY OF CANCER.

VARIOUS reports appearing in the literature from time to time have given the impression that we are on the threshold of success in the search for a chemotherapeutic cure for cancer, and it is cheering to read: "I have no hesitancy in expressing the opinion that cures for the many different kinds of cancer affecting man will be achieved by the use of chemical agents alone." These were the concluding words of the Nathan Lewis Hatfield Lecture for 1955,[1] delivered to the College of Physicians of Philadelphia by Sidney Farber, who is, among other things, the Director of Research at The Children's Cancer Research Foundation, Boston. In the course of his lecture Farber bypassed surgery and radiology, which cure cancer by removal or physical destruction, although he did mention the action of chemical agents which render the normally irradiation-resistant rhabdomyosarcoma sensitive to irradiation, and the part played by chemicals in conjunction with irradiation in converting inoperable rhabdomyosarcomata and neuroblastomata into operable ones. Farber also limited his discussion of the chemotherapy of cancer by making only passing reference to the action of hormones, whose effects on some types of cancer are unsurpassed by any other form of chemotherapy; he did point out, however, that efforts are now being made to synthesize analogues of œstrogens and androgens with the aim of increasing carcinolytic powers while decreasing undesirable side effects. Two other classes of chemotherapeutic agents which are showing great promise, but which have achieved nothing definite yet, are plant extracts such as podophyllin and its derivatives, α and β peltatin, and bacterial products such as certain polysaccharides. Two of the antibiotics, puromycin and its derivative 4-aminonucleoside, and actinomycin D, have had their carcinolytic action against certain mouse cancers definitely demonstrated. These have no destructive effects on bone marrow, in man as well as in the mouse, although they have so far had no reported effect on human cancer. Destruction of bone marrow is the difficulty with the cytotoxic group, together with other toxic effects of less importance. However, dihydrotriazines which do not affect bone marrow have now been synthesized. Of this group, the nitrogen mustards have the power of cross-linking proteins, thus immobilizing cell function; they also bind sulphhydryl enzymes, thereby blocking respiratory and oxidative function. The mustards also cause depolymerization of desoxyribose, thus interfering with the nucleic acids of the chromosomes and preventing mitosis. Finally, there are the antimetabolites, of which the antagonists to folic acid, the citrovorum factor, riboflavine or pyridoxin, and the antagonists to purines, pyrimidines, thiamine and amino acids are the most promising. Antimetabolites represent, according to Farber, some of the most rational chemotherapeutic agents; their value, however, must depend on two factors: first, the proof that there is some difference between the nucleic acid metabolism of normal and of cancer cells, and secondly the finding of selective nucleic acid antagonists to exploit this difference. As yet unexplored are similar antimetabolites which may affect other essential cell constituents, such as lipids, carbohydrates, minerals, or even water.

In order to achieve further results, Farber points out that many thousands of substances will have to be screened, chiefly on a wide range of mouse tumours, since no experimentation, in the laboratory term, may be exercised on a human patient—nothing may be done except for the benefit of the patient. Nevertheless, since it has been found possible to transfer certain human cancers to the cheek pouch of hamsters or to rats which have been pretreated with cortisone or irradiation, it is possible that various chemicals can be tried on a series of such transplanted tumours from a patient with disseminated and inoperable cancer, in the hope of finding a chemical that is effective.

Farber explained that screening of chemotherapeutic agents against cancer is going on in many parts of the world, and that it is most important that screening programmes be correlated and information be rapidly disseminated throughout all centres as rapidly as possible. Methods for doing this are now being developed by the Committee on Chemotherapy of the National Advisory Cancer Council; it is to be hoped that cooperation in these matters will be forthcoming, and that by such cooperation these promising methods of research will be accelerated and soon brought to a successful end.

[1] *Tr. & Studies Coll. Phys. Philadelphia*, August, 1955.

EMERGENCY SUTURING.

A NOVEL METHOD of suturing when fine surgical needles are not available for securing neat cosmetic results, or even when no ordinary surgical needles are available, is described in the *Medical Technicians Bulletin* (U.S. Armed Forces) of July-August, 1955. Fine hypodermic needles are inserted through both edges of the wound, then threaded with fine suture material, or even with their own stylets. The needles are then withdrawn, leaving the sutures in place ready for tying off or twisting up. This is undoubtedly a clever trick, worth recording. It may help someone in an awkward situation.

Abstracts from Medical Literature.

OBSTETRICS AND GYNÆCOLOGY.

Carcinoma of the Ovary Following Hysterectomy.

V. S. COUNSELLER, W. HUNT and F. H. HAIGLER (*Am. J. Obst. & Gynec.*, March, 1955) review a series of 67 cases of carcinoma of the ovary, occurring in women who had previously undergone hysterectomy for a benign condition. When hysterectomy was performed only two patients were in the age group thirty years or less, 20 were in the group thirty to thirty-nine years, and 45 were aged forty years or more. When the carcinoma was found, only two patients were less than forty years of age, whilst about 70% were in the age period from forty to fifty-nine years. Thus the cases tended to be closely related to the period of the menopause and involution of the ovaries. In about 80% of the cases the interval of time between the hysterectomy and the subsequent discovery of the carcinoma was five years or more. The five-years survival rate for 44 traced patients was 36·4%. The authors state that the fact that patients forty years or older in the series represented 67·1% of those who underwent hysterectomy but 95·5% of those with malignant disease of ovaries makes it seem reasonable to advise castration with hysterectomy in that age group. On the other hand, it seems doubtful whether patients less than forty years of age who are to have a hysterectomy for a benign condition should be advised also to have castration to prevent subsequent carcinoma of the ovary.

Diagnosis and Treatment of Endometriosis.

J. P. PRATT (*West. J. Surg.*, April, 1955) discusses practical problems in the clinical diagnosis and treatment of endometriosis. He considers that the condition is now being better diagnosed rather than increasing. The diagnosis may be made pre-operatively, at operation or by the pathologist. A correct pre-operative diagnosis in 75% of cases is considered by the author to be a good record. The condition of endometriosis is neither a tumour nor an inflammatory lesion, but a growth of tissue like the endometrium growing elsewhere than as a lining for the uterine cavity. The author stresses the fact that endometriosis is subject to ovarian control within the limits imposed by the tissue of its adoption. Internal endometriosis or adenomyosis is thought to arise from an extension of the uterine endometrium. Menorrhagia is an important symptom, and pain is not a feature of this condition. Diagnostic curettage may reveal scanty endometrium in contrast to conditions such as fibroid tumours, endocrine hyperplasia and malignant disease. Ovarian endometriosis may vary considerably in degree from microscopic spots to large chocolate cysts. Suggestive symptoms are pain, dyspareunia and a sense of pressure in the pelvis. There is a cyclical fluctuation of the pain and of the tenderness and size of the pelvic mass. Inflammatory lesions of the adnexa are usually more acute in onset and are more tender on bimanual examination than is the case in endometriosis. The presence of fever and leucocytosis is an aid in the differential diagnosis. No treatment is indicated for small endometriotic lesions of the ovaries, and the management of extensive lesions is highly individualized. When treatment is necessary, surgery is preferred to hormonal or radiation therapy. When necessary, it is possible to remove one ovary and half of the other ovary without appreciable impairment of fertility. The author briefly discusses endometriosis of the uterine tubes, round ligaments, pelvic peritoneum, utero-sacral ligaments, recto-vaginal septum, bowel, bladder and laparotomy scars. He states that many of these varied lesions require no treatment, conservative surgery is practised when possible, and when operation is necessary the reduction of ovarian function by the excision of one ovary and half the other ovary has had satisfactory results. The author concludes that the management of patients with endometriosis remains highly individualized. Whatever the location of the lesion its activity depends on ovarian function. Recognition of this relationship is essential to correct diagnosis and intelligent treatment.

Female Genital Tuberculosis.

B. P. ZUMMO, H. SEBED AND F. H. FALLS (*Am. J. Obst. & Gynec.*, July, 1955) present a study in diagnosis and prognosis of 64 patients with genito-peritoneal tuberculosis from the Cook County Hospital. They state that it is generally agreed that 5% or less of all disease of the Fallopian tubes is due to tuberculosis, as are 1% to 6% of all sterility problems. The diagnosis of tuberculosis may be most difficult because of the way in which it masquerades as other gynæcological conditions. The incidence of missed pre-operative diagnosis was 30%. The most common conditions mistaken were partial or complete intestinal obstruction, chronic pelvic inflammatory disease, recurrent appendicitis and fibromyoma of the uterus with pelvic inflammatory disease. Of the 64 patients, 25 had pulmonary involvement and 10 had gastro-intestinal abnormalities suggestive of tuberculosis. Endometrial and cervical histological biopsies are useful procedures—34 positive results were obtained in 64 cases—and are complementary to bacteriological studies. Hæmatological studies showed a relative or absolute leucopenia, and at least 50% of patients with active infection will have abnormally low hæmoglobin levels. Before the advent of antituberculosis chemotherapeutic and antibiotic drugs (1949), of 28 patients treated 18 died, four were alive, and six were untraced—over a period of three years. The combination of streptomycin with PAS and isoniazid has dramatically changed the prognosis. "Silent" lesions in the endometrium and oviducts respond promptly, and pregnancies are now known to have occurred provided the pathological changes were not too advanced. Many patients have been spared the ordeal of surgery, although adnexal masses, abscesses, prominent thickenings, infiltrations and ascites in many instances still require surgical treatment. Operative and post-operative hazards are reduced.

Management of Mitral Stenosis in Pregnancy.

C. L. MENDELSON (*Am. J. Obst. & Gynec.*, June, 1955) presents the results of cardiac surgery in relation to pregnant women at the New York Lying-In Hospital. Over a period of twenty years, 29 deaths have occurred in 2932 patients diagnosed as having heart disease—a mortality rate of 1%, or 23% of all deaths at the institution. In 90% of cases the disease is of rheumatic ætiology, and approximately 75% of the patients have mitral stenosis. A review of the literature shows that 16 patients have undertaken pregnancy subsequent to valvulotomy, and all went through pregnancy without cardiac difficulty. Forty patients had the operation *ante partum* at times varying from the second to the thirty-sixth week of gestation. All but two survived; one had to have an abortion, and the remaining 37 did well. Pregnancy does not increase the operative mortality, and the danger of producing premature labour is not significant. The indications are that mitral commissurotomy prior to and during pregnancy favourably influences maternal and fœtal mortality. The author concludes that cardiac surgery has made child-bearing possible for women heretofore doomed to barren marriage by severe mitral stenosis.

Adenocarcinoma of the Endometrium.

G. A. WEBB, A. J. MARGOLIS AND H. F. TRAUT (*West. J. Surg.*, July, 1955) report a clinico-pathological study of 261 cases of adenocarcinoma of the endometrium. They attempt to evaluate factors influencing the prognosis of this disease and outline a plan of treatment based on these factors. They note a generally increased incidence of endometrial carcinoma and an alteration in ratio to squamous carcinoma of the cervix from 1 : 6 to 1 : 3 in their clinic during the past fifteen years. Two factors are suggested as of possible significance in the increased numbers of fundal cancer: the increased longevity of the population and the use of œstrogens in an exceedingly large number of the aging female population. The five-year survival rate of this series has remained stationary at 51% since a previous report from the same clinic in 1937. A clinical classification of patients is made into seven stages, and an histological classification is made comprising four types. The five-year and ten-year survival rates from these figures indicate that the deeper the myometrium is involved, the less is the chance of survival. When deep myometrial invasion has occurred, the tumour cells have usually spread beyond the uterus. The prognosis is progressively worse with the degree of anaplasia of cancer cells. The prognosis of patients with adenoacanthoma was similar to that of patients with papillary adenocarcinoma of intermediate differentiation. The authors evaluate treatment in the series of cases under the following headings: surgery alone, radium and

surgery, radium and surgery *plus* X rays, surgery and X rays, radium alone, radium and X rays. The five-year survival rates for operable cases showed no significant difference according to the method of treatment. On the contrary, the ten-year survival figures showed that surgery, combined with radium or X rays, gave the best results. On the basis of these observations the authors propose a plan of therapy for endometrial carcinoma which may be summarized as follows: (i) In the presence of a small uterus and a well-differentiated growth pan hysterectomy is adequate; if deep myometrial invasion is found, additional X-ray therapy is indicated. (ii) A small uterus with poorly differentiated adenocarcinoma is treated by the application of radium followed by panhysterectomy six weeks later; if deep muscle invasion is present or lymph nodes are found to be involved, deep X-ray therapy is given post-operatively. (iii) A large soft uterus with a well-differentiated type of neoplasm is treated by preliminary radium application, followed by a Wertheim hysterectomy; X-ray therapy is again given if lymph nodes are involved. (iv) A large soft uterus with a tumour which is poorly differentiated or which is adenoacanthomatous and any endometrial carcinoma which has spread to the cervix are treated by the sequence, radium application, Wertheim hysterectomy and X-ray therapy.

Surgical Treatment of Prolapsus Uteri.

H. C. STEARNS (*West. J. Surg.*, July, 1955) discusses various operative procedures for the cure of genital prolapse and reports results following vaginal hysterectomy, Manchester operation and the Le Fort operation. The anatomy of the uterine supports is briefly outlined. The ætiology of prolapse is considered by the author to be based on three important factors: constitutionally inadequate supporting tissues, age with associated atrophy of the supports and the trauma of childbirth. He states that no single operative procedure may wisely be applied to all patients with prolapse. The patient's age, her health and the desire for more children, as well as the presence of coexisting pathological changes and the severity of relaxation, have important bearings on the choice of operation. Of 202 reported cases of prolapse, 159 were treated by vaginal hysterectomy, 35 by the Manchester operation and eight by Le Fort's operation. The advantages and disadvantages of each operation are listed, and the author concludes that vaginal hysterectomy takes precedence over all other operations in the treatment of uterine prolapse. He employs this operation in all cases of prolapse in which the quota of desired children is filled, the patient offers a good operative risk and the performance of the operation is expected to rectify the condition permanently. The Manchester operation is chosen in younger patients when child-bearing function is desired and in older patients when a shorter operation is preferred to vaginal hysterectomy. The author states that post-operative delayed hæmorrhage, pyometra and the risk of subsequent neoplasm of the uterus are possible complications and disadvantages of the Manchester operation. The Le Fort operation is thought to have a limited but valuable place in the correction of prolapse. The author prefers this operation in elderly and feeble patients who have passed the stage of sexual activity and whose physical condition demands a short operation. He considers that the Manchester operation has in most cases superseded the Le Fort operation, but the latter operation is advantageous in patients with extensive inversion of the vagina following total hysterectomy. The essential features of pre-operative and post-operative treatment are stated, and the necessity is stressed for correcting any existing or incipient enterocele.

Chlorpromazine and the Obstetric Patient.

M. KARP, V. E. LAMB AND H. B. W. BENARON (*Am. J. Obst. & Gynec.*, April, 1955) submit a preliminary report on the treatment of nausea and vomiting in labour by the drug chlorpromazine together with the effects of this drug on analgesia and anæsthesia of delivery and the effects on the newborn baby. They state that anæsthetists have observed that chlorpromazine has a hypnotic and sedative action on the central nervous system and a potentiating effect on analgesics and anæsthetics. Chlorpromazine was administered to 114 obstetrical patients; 100 patients were untreated and served as a control group. A dosage of 25 milligrammes was given to patients in active labour and repeated after four to six hours, depending on the state of sedation. The patients were treated by chlorpromazine alone or in combination with meperidine, 50 milligrammes, and scopolamine, 0·4 milligramme. The anæsthesia for delivery consisted of inhalation anæsthesia in 75 cases, spinal anæsthesia in 24 cases, pudendal block in nine cases, and no anæsthesia in six cases. The incidence of nausea and vomiting during labour was 16%, in contrast to a total incidence of 27% in the control group. The authors consider that both the incidence and severity of nausea and vomiting during labour were reduced by chlorpromazine. Moreover, the drug had a sedative and hypnotic action *per se*, and all combinations of chlorpromazine and analgesics without scopolamine were more effective than when the analgesics were used alone. The authors noted a "medical prefrontal lobotomy" effect in some patients, characterized by torpor, a mask-like facial expression and a quiet phlegmatic acceptance of pain. The height of analgesic and sedative effect of chlorpromazine was noted one and a quarter to two hours after intramuscular injection of 25 milligrammes. Chlorpromazine did not appear to exert any significant effect on the duration of labour. However, the strength of uterine contractions was occasionally reduced in *primigravidæ*. Two patients had inertia following delivery and developed excessive bleeding. No harmful effects of the drug on mother or child were observed in the series of cases. Certain side effects, such as flushing, dry mouth and flaccidity, were noted in the treated patients, and a hypotensive effect with an average drop of 10 millimetres of mercury in systolic and diastolic blood pressure was observed. A scheme of dosage in combination with other sedatives is suggested by the authors.

The Third-Degree Laceration.

R. W. FULSHER AND C. L. FEARL (*Am. J. Obst. & Gynec.*, April, 1955) report and review 37 third-degree perineal lacerations which have occurred at Emmanuel Hospital during the past five years. The incidence of these lacerations was 0·42% with median episiotomies and 0·08% with medio-lateral episiotomies. Three of the complete tears were associated with spontaneous delivery, 28 with elective use of low forceps, five with mid-forceps rotations and extractions, and one with a difficult breech delivery. Spinal anæsthesia was used in 19 cases and general anæsthesia in 17. The authors consider that these figures tend to show that third-degree lacerations are more frequent when spinal anæsthesia is used. The average duration of hospital stay for the group was seven days. Sixteen infants in the series weighed more than eight pounds, but the infants weighed less than seven pounds in eight deliveries. Prophylactic antibiotics were used in two-thirds of the cases. The effect of antibiotics and chemotherapy could not be evaluated, as there was no febrile morbidity in the series. During the past three years median episiotomy has been preferred to medio-lateral episiotomy at the hospital on account of easier repair, less bleeding and less pain during the puerperium. The authors consider that medio-lateral episiotomy is indicated when prompt and hurried repair is desirable, in face and brow presentations and in occipito-posterior deliveries. An important part of the episiotomy consists in prolonging it inside the vaginal floor as far as necessary. The authors advise immediate and adequate repair of third degree lacerations according to the following technique: The rectum is stitched with submucosal interrupted chromicized sutures, the mucosa being everted into the lumen of the bowel. A second row of interrupted sutures is then inserted over the first one. The torn ends of the external sphincter are identified, and the thick fascial layer around the sphincter is approximated with interrupted chromicized sutures. No sutures are placed through the sphincter muscle. The rest of the episiotomy is closed in layers in the usual manner. The bowels are not confined after repair of a third degree laceration. An anti-constipation diet and bulk laxatives may be given after the first post-partum day, and perhaps an enema on the fourth day.

Osteoarthropathy and Carcinoma.

F. HARPER AND L. PATTERSON (*Arch. Surg.*, May, 1955) state that osteoarthropathy with painful joints and clubbing of the fingers may occur from two to thirty-six months before symptoms and signs of carcinoma of the lung occur. If the occurrence of pulmonary osteoarthropathy makes the diagnostician direct his attention to the lung, many operable carcinomata may be discovered.

On the Periphery.

MEDICO-HISTORICAL RELICS AT OXFORD AND KENSINGTON.

COUNTLESS SCHOLARS have passed along Oxford's old-world High Street as it wends its way through many of the colleges of the University from Magdalen Bridge, past the Mitre Inn, towards the Norman castle tower beyond its western end. Countless, indeed, are the famous doctors which the "city of spires" has seen in the seven centuries separating the lives of John of Gaddesden and William Osler, albeit but the fleeting glimpse of student years in many instances. Notable evidence of the lives of two practising physicians remains: the Holbein portrait of Thomas Linacre, founder and first President of the Royal College of Physicians, at All Souls College, and the Infirmary which bears the name of John Radcliffe. As befits a university the history of which is so much a part of the history of science, it is the scientific rather than the clinical aspects of medicine to which Oxford has contributed most in the past; the brilliant seventeenth century researches of Robert Boyle, Robert Hooke, Richard Lower and John Mayow are as significant to the science of physiology as the discovery of penicillin is to the new science of therapeutics. To all the scientists who have worked at Oxford there is a permanent tribute, an undedicated but nonetheless true memorial, the Museum of the History of Science. The displays devoted to anatomy, medicine, surgery and pharmacy, with which we are primarily concerned, are unsystematic and incomplete, forming as they do but a small fraction of the scientific treasure exhibited. In themselves, however, they amply repay a visit to the building, which, when erected in 1683, included the first university chemical laboratory in England, "perchance one of the most beautiful and useful in the world, furnished with all sorts of furnaces and all other necessary materials", according to a contemporary account.

The Ashmolean Museum, as the building was originally named, lies in the very heart of Oxford, wedged as it were between the Sheldonian Theatre, the Bodleian Library and the Divinity School, across Broad Street from the grandeur of Trinity College and the glamour of the New Bodleian. In the past century or so the Museum, in outline perhaps the work of Christopher Wren, slowly lost its several functions in regard to the teaching and study of the natural sciences, and it is only in the last three decades that it has regained its earlier importance through its development as the home of an historical scientific collection. A small series of natural history exhibits forms a link with the Museum's foundation, for they come from one of the earliest English scientific collections—more precisely perhaps a collection of curiosities, which could be seen by the public at Lambeth a few centuries ago—built up by the Tradescants, father and son. From them it passed to Elias Ashmole, who presented it to the University, where it occupied the upper floor of the new building in 1683. Alas, the remains of the famous dodos are no longer to be seen there.

At present the upper floor houses a magnificent collection of astrolabes, among a great variety of instruments relating to time-telling, navigation and astronomy. Other exhibits are concerned with mathematics, pneumatics, thermometry and electricity, the latter including some early X-ray tubes and the apparatus used by Sir William Crookes to demonstrate the properties of cathode rays before the Royal Society in 1870. The ground floor is devoted to astronomical instruments, to photography and in particular to microscopy. The collection of microscopes is that of Reginald Clay; the collection of T. H. Court, his collaborator in their classic monograph on the history of the microscope, is in the Science Museum at Kensington. Both are remarkable for their completeness and the quality of the examples shown. The latter collection, incidentally, includes two microscopes used by William Withering. On the same floor some early ophthalmoscopes and pairs of spectacles, including two examples of the latter from the seventeenth century, are to be seen.

Medical history is illustrated downstairs in the old laboratory. The most striking exhibit, appearing so strangely out of place, is a somewhat boat-shaped cane chair on the cushion of which are worked the letters "E.J.": it belonged to Edward Jenner. Among the personal relics of interest are the quarter-minute pulse glass used by John Coakley Lettsom, William Osler's sphygmomanometer (an aneroid type by Verrin of Paris) and Sir Archibald Garrod's "bent-shank" clinical thermometer. Other items of medical apparatus worthy of note are a Marey's sphygmograph, which forms an important landmark in the history of physiological and clinical recording instruments, an excellent set of silver-mounted artificial leeches with other cupping and bleeding paraphernalia, an early Oxford vaporizer, and, in the same vein although of a different era, a complete apparatus for the resuscitation of the apparently drowned (including bellows for artificial respiration via the nostrils and an attachment for the rectal insufflation of smoke). Of the surgical instruments, the perfect case of trephining instruments of the eighteenth century, a seventeenth century mechanical artificial hand, a set of the flexible metal bougies introduced by W. Smyth, and the few, but well-preserved, Roman instruments are remarkable. In the dental section are two eighteenth century pelicans, some excellent pocket sets of instruments, and, as a curiosity, a solid ivory denture.

The history of pharmacy and *materia medica* is well illustrated. A most important and instructive exhibit may perhaps be included in this category—the selection of improvised apparatus (outstanding among which are a modified bedpan and a tin of disinfectant) used by Sir Howard Florey and his associates in early work upon the cultivation and extraction of penicillin. Drug jars, drug chests, early patent medicines and homœopathic medicine chests (one example of which contains no less than 112 tiny phials) are well represented, and in many instances the drugs themselves, long since in the therapeutic graveyard, appear quite well preserved.

A final exhibit of medical interest is the fascinating collection of Japanese medical netsuke. Each figure, small but exquisitely carved, illustrates a physical deformity, as lameness, blindness, external tumour, the various techniques of massage, or the process of moxibustion (a form of superficial cauterization derived from China centuries ago and still widely practised by all classes of Japanese for almost any complaint).

It is perhaps relevant to refer briefly to several medical items in the Science Museum at Kensington, where, of course, they form no specific section, but where they occur occasionally, usually to illustrate some point of scientific progress. A somewhat incongruous exhibit is George Stephenson's silver lancet case, displayed with other personal relics amongst a variety of steam engines; one of the engines, incidentally, had been used by a firm of chemists and druggists for nearly a century (1797-1885). The gallery devoted to acoustics illustrates the development of electrical hearing aids. There also are some beautifully constructed ear trumpets of horn, shell and pewter, some further examples of which are to be found in the comprehensive collection of scientific instruments which belonged to King George III (1760-1820). Traube pattern clinical thermometers, thermometers adapted for physiological research as suggested by Virchow (characterized notably by their length, shape and scale arrangement for ease of use in various situations) and two earlier mid-nineteenth century skin and ear thermometers of mercury type for clinical use are exhibited in relation to the measurement of temperature. The gallery assigned to the science of optics contains a small collection of spectacles, the earliest specimens dating from about 1750. Early examples of rimless spectacles, introduced in about 1825, and of opaque lenses with a small central hole for use in cases of squint (*circa* 1800) are shown. Other ophthalmic apparatus includes notably an excellent example of Perrin's artificial eye and of Couper's optometer, the latter most elaborately made by Curry and Paston in about 1890; the history of the ophthalmoscope would make a valuable addition to this section. Of particular interest is the first X-ray picture taken in England, which is shown in conjunction with a series of exhibits dealing with historical aspects of electrical discharges in gases and the development of the X-ray tube. In fact, the list of items of more or less medical significance could be expanded by reference to almost any of the galleries; there is the apparatus used by Graham a century ago in his work on diffusion and osmosis, an eighteenth century pharmaceutical furnace, a Persian apothecaries' balance and a mortar and pestle or two from the chemist's shop of John Walker in Stockton-on-Tees, the inventor of the friction match in 1826. Those who tour the full-scale representation of a coal mine in this remarkable museum may see the naked candle and wick lamps used by miners of Roman times as well as a number of models designed by Humphry Davey.

Somewhere it has been remarked that an unsuccessful museum may be eloquent of the past, offer a passing message for the present and hold out small hope for the future. Nowhere could the success of a museum be better illustrated than in the two we have chosen to describe; one in the

scholarly quiet of a university, the other amidst the hustle and bustle of a busy metropolis; the one illustrating more perhaps the development of man's thoughts, the other of his practice, both uncompromisingly renewing hope in the future by a contemplation of the achievements of the past.

BRYAN GANDEVIA.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on June 21, 1955, the medical superintendent, DR. NORMAN H. ROSE, in the chair. The principal speaker was DR. J. E. BLACKMAN.

Clinical History.

The following clinical history was presented.

A married woman, forty-five years of age, was sent to hospital with "hepato-renal" failure. Ten days before admission to hospital she suddenly developed a severe pain in the lower part of her abdomen on the right side, a high temperature, vomiting and diarrhœa. The doctor who was called to see her at 5 a.m. prescribed "sulpha" tablets. The diarrhœa stopped, but for the next ten days she had persistent, though diminishing, epigastric and periumbilical pain, and she vomited everything she drank and ate nothing.

On the second day of the illness she became jaundiced. Then her output of urine decreased, and she passed none on the seventh and eight days. For four days prior to admission to hospital she was only half-conscious and her muscles were twitching.

There was no history of contact with jaundiced persons, and she had had no injections or transfusions. Her gall-bladder had been removed two years before her admission to hospital. She had also been under treatment for an ulcer of the stomach. She was a teetotaller.

On examination on admission to hospital she was thought to be drowsy, worried and slightly jaundiced but not dehydrated. Her breath was foul and her tongue coated. Her liver was palpable and tender, some ascites was thought to be present, and her urine was dark brown in colour, of specific gravity 1·012 and neutral in reaction, with no evidence of albumin, sugar, bile salts or bile pigments. She passed a loose brown motion.

No other abnormalities were detected in the abdomen, chest or nervous system. The heart appeared normal, the blood pressure was 130 millimetres of mercury, systolic, and 90 millimetres, diastolic, and the pulse rate was 80 per minute. She was afebrile.

The patient was given various fluids intravenously and a carbohydrate-fat diet through an intragastric tube. No urine was passed on the first day, two and four ounces were obtained by catheter on the second and third days, then she wet the bed on the succeeding two days. On the sixth day she passed only one ounce, her condition deteriorated, and she died next day.

On her admission to hospital the results of special investigations were as follows: the hæmoglobin value was 6·8 grammes per 100 millilitres of blood; a microcytic hypochromic anæmia was present with a neutrophile cell leucocytosis and plenty of platelets; a plain X-ray examination of the abdomen showed no abnormality; the total serum protein content was 5·9 grammes *per centum* (albumin 3·7, globulin 2·2 grammes *per centum*); thymol turbidity was three units; the alkaline phosphatase content was 11 King-Armstrong units; the serum bilirubin content was 0·5 milligramme, the blood urea content was 277 milligrammes and the blood creatinine content 6·5 milligrammes per 100 millilitres; the serum electrolyte contents were: of sodium 119 milliequivalents and of potassium 8·3 milliequivalents per litre; the carbon dioxide combining power of the blood was 26·3 milliequivalents per litre. On the next day, after having received 5% glucose and glucose-saline solutions intravenously and intragastric feeding (Bull's regime), her condition was not changed, and the electrolyte contents were: sodium 115 milliequivalents, potassium 7·95 milliequivalents, chloride 68 milliequivalents and carbon dioxide 18·7 milliequivalents per litre. Later she was given 200 millilitres of 25% glucose solution intravenously and an intramuscular injection of 25 units of insulin. Five hours later the electrolyte contents were: sodium 110 milliequivalents, potassium 7·4 milliequivalents, carbon dioxide 16·5 milliequivalents per litre.

On the third day her level of consciousness had improved, but her general condition was poor, and she appeared to be dehydrated. Her electrolyte levels were practically unchanged, and she was given 500 millilitres of normal saline intravenously and a blood transfusion. She had difficulty in breathing and swallowing, and she had diarrhœa. Sodium bicarbonate was given through the Ryle's tube, 18 grammes in four hours.

Her condition improved, and next day she wet the bed. Her electrolyte contents were then: sodium 133 milliequivalents, potassium 5·7 milliequivalents, chloride 64 milliequivalents and carbon dioxide 16 milliequivalents per litre. Bull's regime was continued, together with five ounces of water by mouth and more sodium bicarbonate.

It was on the sixth day that her condition deteriorated, her pulse became rapid and thready, and her blood pressure fell. The serum sodium content was 135 milliequivalents, potassium 3·85 milliequivalents, chloride 66 milliequivalents and carbon dioxide 12 milliequivalents per litre; the blood content of urea was 276 milligrammes and of creatinine eight milligrammes per 100 millilitres; the serum calcium content was 6·6 milligrammes per 100 millilitres. She was given potassium chloride, "Bovril" and orange juice by mouth, saline, glucose and Darrow's solution intravenously, calcium gluconate intramuscularly and injections of methedrine and later nor-adrenaline. She was cold, sweaty and tetanic. Her blood pressure increased, but was not long sustained, and her temperature rose to 104° F. On the day she died her serum electrolytic contents were: sodium 141 milliequivalents, potassium 5·78 milliequivalents, chloride 88 milliequivalents and carbon dioxide 10·7 milliequivalents per litre.

The normal electrolyte values are: sodium 135-150 milliequivalents, potassium 4-5 milliequivalents, chloride 95-105 milliequivalents and carbon dioxide 25-33 milliequivalents per litre.

Clinical Discussion.

DR. NORMAN H. ROSE: Today we are met to hear Dr. Blackman discuss a patient of unusual interest. I introduce Dr. Blackman to you.

DR. J. E. BLACKMAN: This evening we have to discuss a patient presenting rather unusual features, who was admitted to the hospital and died some few days later. It is to be noted that she was a married woman, forty-five years of age. Ten days before admission she had severe onset of lower abdominal pain, vomiting, diarrhœa and fever. Jaundice was noted on the next day, and it was not until the eleventh day, on her admission to hospital, that we knew anything about her physical signs. We know that during this pre-admission period she had a decreasing urinary output, and that on the seventh and eighth days she passed no urine at all. We are left in doubt as to the ninth and tenth days; we know that on the twelfth and thirteenth days she passed two and four ounces respectively. On the fourteenth and fifteenth days she had a wet bed, an unknown quantity of urine being passed, and on the sixteenth day, the day before death, she passed only one ounce.

This patient presents, therefore, with renal failure. From the outset one would like to consider whether this was acute renal failure *per se*, or an acute episode of a chronic illness. In this regard we would like to know something of her past health, whether she had had a urinary tract infection, nephritis, pyelonephritis, obstruction or operation on the urinary tract. She was a woman of forty-five, and therefore it is important to know something of her menstrual history, whether she was pregnant at the time and whether she had any history of abortion. These are all pertinent points which are not available. We have nothing to suggest that this was a chronic lesion, and I submit that this patient had acute renal failure.

I should like to attack the problem from this point of view. If she had acute renal failure, then we must postulate either renal circulatory insufficiency, some organic damage to the kidneys, or an obstruction to the urinary tract. I would like to take the point of obstruction first, because this can generally be ruled out by the passage of ureteric catheters. This was not done; so exclusion of obstruction is not so easy. If we postulate that this was due to obstruction, lesions which would occlude both ureters suddenly, such as tumours in the pelvis, come to mind. Have we any details of pelvic examination?

RESIDENT MEDICAL OFFICER: No pelvic examination was done.

DR. BLACKMAN: This is important because, not only from the point of view of tumours, but infections involving the

pelvis could perhaps have been detected. Bilateral ureteric obstruction is reasonably common in advanced carcinoma of the cervix, but I feel this patient does not have features suggestive of that type of lesion. Stones in the urinary tract do not commonly cause obstruction to both ureters, but it does sometimes occur; and this patient presented with pain in the right iliac fossa, and it could easily be suggested that she had a stone in the pelvic ureter on the right side. However, one causing complete, or almost complete, obstruction would be expected to produce some pain in the loin. Also there was associated fever suggesting infection, but we find there are no pus cells in the urine, and I feel that stone is unlikely. A plain X-ray examination was made to exclude stones. Could we see the film?

As far as one can see there is no obvious shadow suggesting stone in the urinary tract, but one could not positively exclude it. The other thing of importance is that the stone might be transradiant, and a plain film would not show it. A transradiant stone could easily have obstructed the ureter of a single functioning kidney or of a pelvic kidney. The renal shadows are not well shown, and the kidney may have been pelvic in position, and a stone could be overlying the dense bony shadows of the sacrum.

Therefore, I do not think we can, on the evidence, completely rule out obstruction, although I think this particular patient did not have an obstructive lesion.

The other diseases which could produce the clinical course taken by this patient are those due to circulatory renal insufficiency causing ischæmia to the kidney, and those causing organic damage to the kidney. Of those causing acute damage I would like, in passing, to rule out acute nephritis. However, I think there is ample evidence in this patient to suggest there are several factors linked together to produce renal ischæmia *plus* toxic changes causing renal damage.

Let me refer again to this first slide, showing the onset with jaundice, decreasing renal function, a period of anuria and then the passage of urine again, which follows very closely the pattern of acute tubular necrosis. We have the stage of onset, the period of anuria and the early diuretic phase in which the patient died. There is severe derangement of renal function. The carbon dioxide combining power is low and progresses downwards with an increasing acidosis. The serum potassium level is initially high, then decreases slightly, probably owing to treatment at this stage. The findings for both serum sodium and serum chloride content were low; some rise of sodium level occurred when sodium bicarbonate was given to the patient. The blood urea and creatinine values were high and remained high throughout.

This picture is very similar to that occurring with acute tubular necrosis following an abortion, as can be seen from the article by Bull in "Recent Advances in Pathology". The rise in blood urea and potassium levels and the low values for sodium and chloride are well shown in relation to the urinary output during the various phases—stage of onset, anuria, early and late diuresis. Renal blood flow falls abruptly almost to nil at the onset and then later gradually increases during the diuretic phase. Tubular function likewise declines to improve only in the late stage of diuresis. Perhaps Dr. Wardlaw would like to comment on the biochemical findings in this patient.

DR. H. S. H. WARDLAW: I do not think I can add much to a very clear exposition given by Dr. Blackman. The blood urea nitrogen content, as he mentioned, was over 200 milligrammes per 100 millilitres, and the blood creatinine content was over six milligrammes per 100 millilitres. The chances of survival with figures like those are supposed to be very remote. As regards the figures for the electrolytes, the sodium level was extremely low, and the rise in potassium content was close to, if not actually at, toxic levels. This was probably partly due to withdrawal of potassium from the cells in response to the low concentration of electrolytes in the circulating fluid. I should like to have known what the reaction of the vomitus was, whether it was acid or not; for apparently the patient was losing very little sodium in the urine, and yet the serum level was very low. Towards the end of the picture the electrolytes were returning towards normal as a result of therapy, but in spite of this the condition of the patient was not improving.

DR. BLACKMAN: Acute tubular necrosis may occur in severe shock, blood loss, severe cardiac failure, dehydration, in various states of poisoning, severe infections, bacterial endocarditis, mismatched transfusion, crush syndrome, to name a few of the seventy odd causes which have been enumerated. We must also add concealed accidental hæmorrhage and septic abortion. This patient being a female, we must consider these factors carefully.

What help can we get from the symptoms? Let us take them in turn. Severe pain—the patient developed sudden severe pain in the lower part of the abdomen on the right side, high fever, vomiting and diarrhœa. She also was under treatment for an ulcer. Was this a perforation? Here we could have shock, fluid loss, vomiting and so on. I do not feel it was. She had no upper abdominal rigidity at any stage. The pain began in the lower part of the abdomen and then spread upwards. A slow leak running down the right paracolic gutter could cause pain in the right iliac fossa, but pain started in the lower part of the abdomen and spread upwards. She had a history of gall-bladder trouble and had had a cholecystectomy. One could wonder whether pancreatitis might have developed, as sometimes happens after removal of the gall-bladder, and whether this caused the shock and jaundice. Again the absence of upper abdominal rigidity and pain would rule this out.

The common cause of pain in the right iliac fossa is appendicitis, but this pain started in the right side without any initiation in the periumbilical region. A pelvic appendicitis developing an abscess or perforating into the pelvis cannot be ruled out lightly, as many of the symptoms point to a pelvic peritonitis. She had severe diarrhœa to begin with; she had a raised temperature and vomiting. Pelvic peritonitis, spreading to the abdomen, would cause lower abdominal pain, which later spread upwards.

A pelvic abscess may have arisen following septic endometritis or salpingitis. Torsion of a tubovarian mass, a large fibroid with infarction or a ruptured ectopic pregnancy must be considered. A ruptured ectopic pregnancy may have caused shock and pain, with blood loss, but I feel the appearance would have been one of pallor and collapse rather than one of jaundice when first seen. Another less likely lesion is endometrioma, causing obstruction to ureters as well.

Another factor to be considered from the history is that the patient was given sulphonamides on the first day. This may have been associated with some other factor, such as an acute intercurrent infection, and in itself could have caused the renal lesion. The patient may have been hypersensitive to sulphonamides and developed an acute tubular necrosis with some blockage of the tubules by crystals. The persistent vomiting could easily have caused an irreversible state of dehydration.

The next big factor we have to consider is the jaundice. On her admission to hospital the serum bilirubin content was within normal limits. Could this then have been something simulating jaundice, perhaps some pigmentation of skin? Then there is the finding of an enlarged tender liver. This is not due to cardiac failure, which can sometimes cause renal damage like this. Is it due to some intercurrent disease such as infective hepatitis? I feel that this is not so. It is much more likely that the jaundice is a hæmolytic jaundice associated with a circulating toxin or some severe infection or poison, which had given her a severe intravascular hæmolysis with jaundice lasting a day or two. I do not think there is any suggestion of obstructive jaundice, as the stools are adequately coloured, the urine contained no bile salts or pigments, and the results of liver function tests appeared normal. Perhaps one of the physicians would give us his view on the liver and the jaundice, Dr. Robertson?

DR. T. I. ROBERTSON: I have very little to add. The results of liver function tests seem to be normal. The total protein is at the lower end of the normal range. The thymol turbidity and serum alkaline phosphatase contents are normal. The serum bilirubin content also is normal. Though most people have only 0·1 to 0·3 milligramme, 0·5 milligramme per 100 millilitres is within acceptable normal limits. I am afraid there is no clue in the data presented here concerning hæmolysis. There is nothing in the blood count or relevant tests which elucidates the problem. We have just a bald statement of the hæmoglobin value and the serum bilirubin content. I cannot help much.

DR. BLACKMAN: The urine showed a specific gravity of 1·012. I was wondering whether this and other tests of urine were repeated, whether 1·012 was a constant finding, and whether at any other time pigment casts, red cells or albumin were found.

RESIDENT MEDICAL OFFICER: Some pus cells, epithelial cells but no crystals were found. The specific gravity constantly was 1·012.

DR. BLACKMAN: The constant, low specific gravity with oliguria indicates that there was severe renal damage. If there had been acute tubular necrosis with formation of blood casts, they may not necessarily have been seen in the urine at this stage, but they may have been present earlier, before admission.

Summing everything up, it seems that this patient was toxic, had an acute tubular necrosis, and the cause of it may have been pelvic peritonitis or abscess. This may perhaps have followed appendicitis with pyelophlebitis; or more likely, I think, though there is no history to support it, she may have had an undisclosed septic abortion or concealed accidental hæmorrhage.

DR. ROSE: Dr. Blackman has very clearly presented a summary of this patient's condition. It does appear that the diagnosis of renal failure has been clinched, but I think there has been insufficient discussion about the cause of this trouble. One would rebuke a casualty surgeon, I think, if he admitted to hospital a patient complaining of acute lower abdominal pain and a full investigation had not been made. Whether information was obtained on initial examination of the patient and it has not been disclosed in the notes I do not know. But we invite discussion on the possible cause, or causes, of renal failure in this case. Mr. Pearson, would you care to say something?

DR. H. H. PEARSON: Would you like me to say something about the management of these patients?

DR. ROSE: I suggest we discuss only the diagnosis at the moment.

DR. PEARSON: I have been associated at one of the women's hospitals with quite a number of cases of this nature, and, to me, jaundice, pelvic pain and acute renal failure spell post-abortal *Bacillus welchii* infection with acute tubular necrosis. Reading through this history, in spite of her age, I think Dr. Blackman has the right diagnosis. In any case she had a pelvic infection, I should think, with a toxin causing hæmolysis and associated with acute tubular necrosis. I can think of no other condition that would give those several findings.

DR. ROSE: We seem to be confirming the diagnosis even further. It does appear now that the renal failure was due to some chemical or bacterial toxin. We have here today one of our foremost toxicologists. Dr. Smith, would you care to say something?

DR. G. C. SMITH: The point which interested me about this patient was whether there was any exposure to, or contact with, some toxic chemical. I would like to know about her occupation, whether she worked in industry at all. But even if she were a housewife, it would still be possible for her to have had some contact with a toxic chemical, because so many of them can be purchased now and are used for such a variety of purposes. Such substances as carbon tetrachloride, used in dry cleaning solvents, and various other types of chlorinated organic compounds—for example, some of the insecticides like DDT, chlordane and the more toxic aldrin and dieldrin—are available to the public. Tetrachlorethane is another very poisonous one. It would be possible for her to have had some contact with one of these chemicals. The chlorinated compounds cause toxic changes in both the liver and the kidney; so that people who are poisoned with, say, carbon tetrachloride may have severe renal damage as well as jaundice. I think, then, that this possibility has to be considered.

RESIDENT MEDICAL OFFICER: I explored that possibility by having the husband prepare a list of all the things used in the laundry and elsewhere about the house. She occasionally used carbon tetrachloride for cleaning. I asked about gammexane and all those related compounds, and I also had him bring in the medicines which she had taken and sleeping tablets.

DR. SMITH: That is very interesting, because certain individuals need only an occasional or relatively small exposure to carbon tetrachloride to cause severe damage. Quite a number of cases have been reported of people having a relatively small exposure to dry cleaning fluid who have been affected and, in some instances, succumbed. If carbon tetrachloride has been swallowed, symptoms may come on as late as ten days after the event; they are not always immediate.

DR. ROSE: We expected you to clarify the situation and not to confuse it. Dr. Jacobs, this is in your department.

DR. L. A. JACOBS: After what Dr. Smith has said *re* poisons, I am not so sure that this is my department! Further, I am now going to be much more careful not to drop food on my clothes, since the use of cleaning fluids can lead to such dire results ten days later.

I do not think that I can add very much more to what Dr. Blackman and Dr. Pearson have said. On the principle that common things are the commonest, one can only put a case like this into the group that one most often sees. For myself this would certainly be the anuria following a septic miscarriage. The symptoms we find here, in chronological order, of a person becoming sick with severe lower abdominal pain, pyrexia, vomiting and diarrhœa mean something amiss in the pelvis; jaundice then developing means that the infection has been severe enough to cause hæmolysis, the whole process then going on to lower nephron nephrosis and renal failure. Whether this was due to toxins, the products of hæmolysis or the sulphonamides she was unfortunately given, I do not know. The fact that she was taking nothing by mouth and was losing fluid by diarrhœa and vomiting is an added factor causing trouble when sulphonamides are given.

I do not think that I can add more. The diagnosis presented seems to me to be a very reasonable one and is in fact the only one which seems to fit into the clinical picture. If we have gone astray, I will be very glad to know where.

Autopsy Report.

DR. A. A. PALMER then presented the following autopsy report.

The body was that of a well-nourished middle-aged woman with post-mortem lividity and *rigor mortis*. A bed sore was present over the sacrum, and the skin around the perineum and *labia majora* was inflamed and thickened with one small area of moist gangrene.

Lungs (aggregate weight 44 ounces; normal 32 to 45 ounces): There was congestion and patchy consolidation of the lower lobes.

Heart (10 ounces; normal 9 to 10 ounces): The myocardium was flabby and the right atrium and ventricle were dilated. The valves, coronary arteries and great vessels were normal.

Liver (62 ounces; normal 50 to 60 ounces): The liver appeared normal in colour and consistency.

Spleen (five and a half ounces; normal five and a half to six ounces). The spleen was firm and appeared normal.

Kidneys (right nine ounces, left 12 ounces, aggregate 21 ounces; normal aggregate 10 to 13 ounces): The kidneys were large and soft. The capsules stripped easily, leaving a mottled, "flea-bitten" surface. There were dark red radiating streaks on the cut surface.

Gastro-intestinal tract: An acute duodenal ulcer was present near the pylorus.

Pancreas: Normal.

Suprarenals: Normal.

Pelvis: A pelvic abscess with three to four ounces of greenish-yellow pus was found in the pouch of Douglas. Both uterine tubes contained pus, and the lateral third of the right tube was dilated. The uterus was slightly enlarged and soft, and a plaque of cream-coloured material was adherent to the endometrium of the fundus.

Microscopic Examination.

Uterus: There is evidence of recent miscarriage, and in the fibrin adherent to the endometrium large Gram-positive bacilli are numerous.

Kidneys: Brown granular casts, some containing demonstrable iron, are present in many tubules. The epithelium of the tubules is often broken up and desquamating. There is patchy infiltration with lymphocytes, plasma cells and polymorphs. These appearances are consistent with lower nephron nephrosis.

Culture from pelvic abscess: *Aerobacter aerogenes.*

Summary.

1. Post-abortal infection: probable *Cl. welchii* endometritis and pelvic abscess due to *Aerobacter aerogenes.*

2. Lower nephron nephrosis probably due to *Cl. welchii* endometritis and resulting in uræmia.

3. Acute duodenal ulcer (probably uræmic).

4. Bronchopneumonia, sacral bedsore, perineal cellulitis.

Pathological Discussion.

DR. ROSE: You now know the diagnosis. We ask ourselves: could anything have been done for this patient? You saw from Dr. Blackman's graphs that a diuresis could be expected about the sixteenth or seventeenth day of the illness. Yet she perished on the seventeenth day. Could she have been carried along till Nature came to her aid? Could we have some discussion on that? It was suggested, Mr. Pearson, that you might say something on that.

DR. PEARSON: I should like to say at the beginning that I doubt very much whether this patient could have been saved. She had been ill for ten days prior to admission with acute renal failure. On admission her hæmoglobin value was 6·8 grammes *per centum*, and I have no doubt that the hæmoglobin value had been in that vicinity since she had her hæmolysis. If we assume that the pathogenesis of renal tubular necrosis is due to renal ischæmia, a circulatory toxin and perhaps obstruction from the products of hæmolysis, then the degree of damage in this particular patient's kidneys at the end of ten days with an anæmia of that severity must have meant that recovery was very unlikely. We see that she did commence to have a diuresis, so it may well be that there were tubules which were recovering but not enough of them to enable her to live.

I was asked to say a few words about the management of these people. I would stress the importance of the first phase, the phase of onset, in relation to the severity of the damage to the nephron. These girls are often admitted in a state of profound shock with a systolic pressure which is almost unreadable. They hæmolyse rapidly, and once this commences they get a profound anæmia. If this is allowed to continue, then the renal damage becomes such that recovery is unlikely. I have seen 15 such cases in the past five years, and with conservative management twelve patients have survived. I think this is attributable to the fact that the moment they are admitted their treatment is regarded as urgent and is started immediately. We try to get their systolic pressure up to 110 or 120 millimetres of mercury, if necessary using a continuous drip with noradrenaline, and we give massive blood transfusions to counteract the effects of hæmolysis. By doing this we have helped prevent further renal damage which would, perhaps, have been irrecoverable. I am sure that this first phase is the important one. So far as the combating of the toxæmia is concerned, they are given 100,000 units of antigas gangrene serum intravenously on admission, a broad spectrum antibiotic and large doses of crystalline penicillin, and the products of conception are evacuated from the uterus as soon as the patient's condition will allow. I think this is a most important phase in the management of such patients; it does not apply to the present case, since she was admitted on the tenth day.

The earlier cases in our series were handled before the hospital acquired a flame photometer, and we did not know a great deal about their electrolytes. Since we have been able to estimate the electrolytes more speedily, the treatment has been along more rational lines.

A restriction of fluids to their insensible loss *plus* their output and a daily estimation of serum electrolytes and carbon dioxide combining power are important. These should be corrected when there is clinical evidence of some abnormality attributable to them. The important ones, I think, are calcium and potassium. Sodium, I have not worried about to any extent. Sodium and chloride always tend to fall with a conservative regime and can be corrected at intervals, but low levels do not seem to disturb the patient. Potassium—I do not quite know how important it is. Of the three patients who died, two died about the tenth day of anuria when they were not uræmic clinically, yet suddenly developed an irregular pulse and died. At that stage we were not doing routine electrocardiograms to determine whether or not there was evidence of potassium intoxication. If the serum potassium content rises to an abnormal level and if at the same time there are any changes in the electrocardiogram, then I think you should take steps to correct it. The best measures are the exhibition of glucose and insulin, as were given here. Glucose immobilizes the potassium in the cell with phosphate glycogen compound, and certainly, as well exemplified here, the serum potassium falls. I have, in three of these people, carried out an exchange transfusion. One embarks on such measures at a late stage as a rule. We have no artificial kidney here. I doubt whether it would prove to be of much value. I say that advisedly because, so far anyway, those that have been produced are very difficult to maintain technically, they require a special team, and all patients with renal failure would have to be sent to the unit. Moreover, when cases are properly managed conservatively, the majority of patients can be saved. Exchange transfusion is an interesting method of removing toxic substances, and diuresis commenced in my three cases within twenty-four hours of such treatment. This also occurred in the case recently reported from Brisbane, where an artificial kidney was used. I do not think that means anything; it just depends on the stage at which treatment is given. Treatment is often given at about the time when a diuresis is going to begin anyway, but it makes one think that there might be something in uræmia from tubular necrosis that prevents recovery of renal function when the tubular epithelium has regenerated.

The third phase, that of diuresis, does not as a rule present much of a problem. There is quite a loss of potassium in the urine, and it is worth having the urinary electrolytes measured at that stage, to watch the degree of excretion. Usually at that stage the electrolytes can be left to look after themselves, provided one administers sufficient fluids and corrects any gross imbalance.

DR. ROSE: Thank you very much, Mr. Pearson. Dr. Blackman, would you care to round off this talk and answer any questions?

DR. BLACKMAN: I think this has been a most interesting case. I would like to thank the various speakers for their helpful discussion.

DR. ROSE: Are there any comments or questions?

PROFESSOR W. K. INGLIS: I should like to put a question to Professor Ward, if I may. If this patient died as a result of what we used to call *Bacillus welchii* infection, is it not surprising that the pigmentation should not have become gradually greater, even after death? Often they have a deep mahogany colour. Secondly, is it not surprising that there was no gas, no gas bubbles, in the liver or in the heart?

PROFESSOOR H. K. WARD: I am no longer a bacteriologist, and I could not have answered that even when I was one. It does seem a pity in the face of an unexplained fever that blood culture was not done. Blood culture may have helped to diagnose the condition early when specific measures could have been taken. We are much too hesitant about the help we can get from a blood culture when there is an unexplained high fever. I am sorry I cannot answer Professor Inglis's questions—but he knew I could not answer them!

What I do not understand is why it was not possible to culture *B. welchii* from the post-mortem specimens. It is a sporing organism and should have been grown in culture.

DR. ROSE: Can anyone answer that, why was the organism not grown?

DR. PALMER: No culture was taken from the endometrium. The culture was taken from the pelvic abscess and I think it is possible that *B. welchii* was not present in the abscess.

DR. ROSE: That would not satisfy you, Professor Ward! I think it has been a very profitable meeting and now it is time to close.

Diagnosis.

Septic abortion leading to renal tubular necrosis and uræmia.

Medical Societies.

PÆDIATRIC SOCIETY OF VICTORIA.

A MEETING of the Pædiatric Society of Victoria was held at the Royal Children's Hospital, Melbourne, on August 15, 1955.

Prophylactic Procedures in the Common Infectious Diseases.

DR. T. F. MCNAIR SCOTT, Director of Research in the Children's Hospital, Philadelphia, United States of America, gave an address on prophylactic procedures in use in the United States of America in the common infectious diseases.

Dr. McNair Scott said that he thought it sometimes worth while to consider what the objectives were in routine immunization procedures. It was clear that when a patient recovered from measles he was immune from another attack because he had developed antibodies against measles. It was recognized that measles swept through the community every few years and affected those who had been born since the previous epidemic. Clinical measles seemed to affect almost 100% of the population. In a recent epidemic of measles in Greenland, where the population was entirely susceptible, the morbidity had been 999 cases out of 1000. However, when one considered some other diseases such as mumps and rubella, it was found that apparently only about 50% to 60% of people had had those diseases by the time they reached adult life; yet epidemics of those infections did not go through the adult population, and they

were no more common than measles. The reason for that obviously was that those particular diseases had infected the population without typical manifestations—in other words, they had caused a subclinical infection. A number of investigators had shown that perhaps 50% of mumps infections were subclinical, and yet after a subclinical infection the patients developed antibodies and were immune to subsequent infection.

Dr. McNair Scott then demonstrated from slides some epidemiological features of measles and mumps. From a study of children in a camp it was found that by the age of fifteen years, 90% of them had had measles, but only 50% had a history of mumps. Other workers investigating a small girls' school had at the beginning of an epidemic of mumps examined blood from the whole school population of 44 and divided them on the basis of their serological reactions into the group that were susceptible and the group that were immune. Only 12 out of 22 of the immune group had a history of mumps; so therefore approximately 50% had had mumps without its being noticed by the parents. They then watched the susceptible group and after the epidemic had ceased retested them, and they found a rise in antibodies in the whole group, whereas only 10 of the 22 had had clinical mumps. That was merely an example of a well-recognized phenomenon. The same situation apparently held true for rubella. There was no pretty example like the one just described, but certainly some experimental workers had shown that it was possible to produce an infection without a rash in a volunteer, and that volunteer was in turn able to produce a full-blown infection in further volunteers. It must be that in these and probably other diseases the resistance of the adult population to infection was maintained by a subclinical infection. Dr. McNair Scott said that it was the objective of immunization procedures to induce such a state of immunity with the minimum of clinical symptoms. That objective fell under the category of active immunization; but as a temporary measure it was possible to use the preformed antibodies of either human or animal origin, and that, of course, was passive immunization. An important modification of passive immunization was what had come to be called passive-active immunization, in which an active immunity was superimposed on a temporary passive immunity. That was the sort of thing that occurred in hepatitis when the individual was given γ globulin but was still exposed to the infection in the community.

Dr. McNair Scott then considered firstly the question of passive immunity, which, he said, was of course immediately available and of value in certain cases. With pertussis it had been found that the blood from human subjects who had been hyperimmunized against the disease contained antibodies which would prevent the incidence of the disease in people exposed if it was given to them. The amount used was 20 millilitres on exposure, in two doses two days apart in infants, and in older children 20 to 40 millilitres. There was also a dried form available, which could be concentrated down to about half that dose given intramuscularly. An even smaller dose could be given when the γ globulin was used, and also an antipertussis serum made in rabbits could be used. Dr. McNair Scott made it clear that he was referring to preparations available in the United States of America, and realized that they might not be available in Australia or applicable to Australian problems.

There was, of course, the well-known tetanus antitoxin that could be given to all immunized persons and used to immunize persons in addition to the booster toxoid dose if the wounds were extensive. Diphtheria antitoxin was also used in a similar way. Toxoid could be given at the same time in a different site and produced an active immunization superimposed on the passive immunization with antitoxin. Penicillin might be just as useful for the prophylaxis of contacts, but active immunization should be started. With passive immunization against measles, to prevent the disease a fairly large dose within five days of exposure was required. To modify measles a much smaller dose as early as possible after exposure was given, and it might have to become larger as the time after exposure was extended. With infectious hepatitis a very small dose seemed to be adequate to prevent at least the clinical manifestations of hepatitis, even though it might not prevent a subclinical infection. With poliomyelitis, 0·14 millilitre per pound was the average dose of γ globulin used in the clinical trials, and that was the dose now recommended. It had been tested out very extensively in a beautifully planned mass immunization experiment in 1951 and 1952. Over 55,000 children were injected, half of them with γ globulin and half of them with an indistinguishable placebo. When the final analysis came, there was a very highly significant decrease in the incidence of paralytic poliomyelitis in those who had had the γ globulin. Further examination in the laboratory had shown that those children who had received γ globulin and did not become paralysed, but were in households in which there was a case of paralytic poliomyelitis, did suffer a subclinical infection as indicated by the finding of virus in their stools and a rise in antibodies against poliomyelitis. So that was probably a valuable tool against poliomyelitis. In the next year when γ globulin was used rather extensively for mass immunization during an epidemic period throughout the United States, a very careful, and as yet unpublished, examination of the results showed a 50% decrease in the incidence of paralytic poliomyelitis in the areas in which it was used.

With mumps a hyperimmune serum had been developed which, if given to an adult who did not know whether he had had mumps or not, but was exposed to his child, might or might not be effective. That was a common problem, and it was difficult to know just what to do about it. However, there were as yet no field trials to show the effectiveness of the procedure.

There was another aspect of passive immunization that was of great importance and played a major role in protection of the newborn. For example, a full-term offspring of an immune mother was not susceptible to measles, mumps and infectious hepatitis, and to *herpes simplex* also if the mother had antibodies against those diseases, as most adults had. It was interesting to speculate why there were exceptions to that general rule. Infants were not immune to various bacterial diseases, and even in certain viral diseases such as varicella they did not seem to be as immune as against measles. Valquist and some of his co-workers in Sweden had shown that all antibodies were not equally transmitted through the placenta, but most of the antibodies bound up with γ globulin went through the placenta. However, typhoid O showed no antibodies in the fœtus, and with *Bacterium coli* there were less antibodies in the fœtus than in the mother. Gamma globulin was not a prophylactic against varicella as it was against measles. Pertussis antibodies on the other hand did seem to be associated with a γ globulin fraction, and yet newborns were relatively susceptible. That might be a matter of dosage, since very small amounts of antibody were necessary when a disease was primarily blood-borne; but much greater amounts were necessary to protect the mucous membranes, which were probably the primary site of infection with pertussis. Infants remained immune for five to six months after birth, in contrast to the passive immunity induced by the physician in his patient, which lasted only three to five weeks with homologous serum antibodies, and two to three weeks with heterologous serum antibodies. Dr. McNair Scott said that the difference between the length of effectiveness of induced and natural passive protection deserved some comment. There appeared to be two factors involved, and one was the dose. Since the level of γ globulin decreased in a linear fashion, the relatively low level of antibody induced by the physician with practical doses would last a shorter time than that transmitted by the mother, who might have suffered from a clinical or subclinical infection and have a high level of antibody. Then, whatever happened to the antibody, whether it was metabolized, destroyed or excreted, the rate of disappearance of antibody in an infant was much less rapid than in an adult. In an infant the half-life was thirty days, which was a decrease of antibody-containing γ globulin of about 14% per week; whereas in the adult, the half-life was twelve days with a weekly drop of 33%. So, of course, whatever dose was given to the baby from the mother, it would take a longer time to disappear than a similar dose induced in an older child or adult. Those were just some points to ponder about.

Dr. McNair Scott then went on to discuss active immunization and said that that was the most valuable approach to protect the community at large. He showed briefly by means of slides the routine procedure in use in the United States of America for immunization of infants. They used a triple vaccine containing diphtheria, tetanus and pertussis. The diphtheria and tetanus toxoids were alum-precipitated and mixed with pertussis vaccine. Immunizations were started at two to three months of age, and two doses were given at monthly intervals with a final dose six to nine months later. He included in the primary vaccinations the dose which was given six to nine months later, and which might be called a booster dose; but it was really part of the primary immunization, and without it there would not be an adequate antibody response. A booster dose of 0·5 millilitre was then given at three to four years of age. There was then some difference of opinion on how much further the triple antigen should be used. Some people used it through primary school up to about ten years, but some,

including Dr. McNair Scott himself, felt that it would be wiser to use the single antigen after that age because of the risk of reaction, which would be referred to later.

The early date of starting immunization was stressed. It had been generally considered that the very young infant did not produce antibodies as well as an older child, and it was also known that, in the laboratory, tissues of the fœtus and newborn animals were extremely tolerant to foreign proteins because they did not produce antibodies. The failure to produce antibodies had been referred to as immunological immaturity. At present it had been considered that the lymphoid tissue seemed to be the site of antibody formation; and therefore in favour of the idea of immunological immaturity was the work of Potter. That work showed that in the newborn there was an absence of germinal centres in the lymphoid tissue and spleen. However, recently the importance of stimulus to the reactivity or the activity of the immunity mechanism had been emphasized. It was known, for instance, that animals who were born in a completely sterile environment produced no lymphoid tissue at any age and had no antibodies, but they did develop it along with antibodies when the stimulus of killed bacterial vaccine was introduced. With regard to infants, Dancis and his co-workers had recently collected some very interesting evidence that the introduction of antigenic stimuli into the environment was more important than the length of maturation of the lymphoid tissue in the infant as a whole. They had injected premature and full-term babies with diphtheria toxoids and found that of 11 premature babies, nine showed no significant response. At birth five full-term infants also showed no significant response, so the difference was not significant. When the premature babies were injected at their estimated date of birth a few weeks later, it was found that out of seven all responded; whereas at the same time in longevity, as it were, the full-term babies did not respond, and the difference was therefore highly significant. That suggested then that it was exposure to an antigen-laden environment that had a greater effect on the antibody-forming mechanism than just time spent in the sterile confines of the uterus. It was only the first attempt at making antibodies that was slow. Once the cells had learned the process of antibody production, then a recall or booster stimulus produced a result as rapid in the young as in the older individual; for that reason Dr. McNair Scott had emphasized the importance of the six to twelve months booster dose.

Dr. McNair Scott said that it would be advisable now to consider in general terms the tools available for inducing immunity in the population and to discuss the pros and cons of their individual use. Since most of the routine antigens fell into the group of killed antigens, he would consider some of the problems involved in using those. The bacterial vaccines in common use, whooping-cough and typhoid, did produce a temporary immunity. That produced by typhoid was short-lived, and revaccination must be done yearly to maintain an immunity; but, of course, that was necessary only for populations at special risk, such as those going into endemic typhoid areas or in epidemic emergencies. Pertussis immunization was at present included among the routine immunizations of infants, and, he thought, quite rightly because of the ubiquitous nature of the infecting organism. It should be mentioned, however, that serious reactions, particularly of an encephalitic nature, could occur and did occur with pertussis antigens; and therefore as a matter of warning, if any child reacted with high fever, especially, and certainly if he had a convulsion following one dose of pertussis vaccine, no further doses of pertussis vaccine should be given. At the moment, with present-day antibiotics, which although not curing whooping-cough did seem to cut down its lethal character, the risk of infection was certainly less than the risk of a serious encephalitic reaction. Booster doses of pertussis vaccine were usually recommended until school age, and some people recommended further doses. However, Dr. McNair Scott thought on the whole that it might be just as well to allow children to take their risk of getting a mild attack of whooping-cough or at least be exposed to the organism in school, so that actually they might produce in that way a more prolonged immunity than with just a repeated series of vaccinations with dead vaccine. If a child had not been immunized in school, it was recommended to give 0·5 millilitre of the saline vaccine subcutaneously three times at three-week to four-week intervals. If a child had been vaccinated and was in intimate contact with a case of whooping-cough, then he thought it was reasonable to suggest a booster dose of the vaccine.

A problem basically of even greater concern was that of diphtheria. Owing to the widespread use of diphtheria toxoid the incidence of diphtheria had been gratifyingly reduced in the population as a whole. However, several studies had shown that the carrier rate of diphtheria bacilli was lower in immunized than in non-immunized populations. That meant that in such immunized populations the number of parasites was so reduced that the host was not constantly being exposed to the natural stimulation so often necessary for continued production of antibodies. Unless therefore those stimuli were repeated artificially, the host after a relatively short time was susceptible to any casual encounter with a virulent organism. It was necessary therefore to be prepared to maintain a programme of artificial stimulation or to deal with a population of susceptible adults; and indeed they were dealing with a population of susceptible adults in the United States of America, and, of course, as a corollary, susceptible newborns and very young infants. Slides were shown demonstrating that, for instance, in Massachusetts in 1954, when all the children were immunized, from the age of twenty years there was a steady rise of people susceptible to diphtheria. In Copenhagen in 1937 and 1944, before the general use of diphtheria toxoid, susceptibles were higher in the young age groups, and gradually the incidence of susceptibles decreased. A slide then showed the routines of diphtheria immunization in the United States of America. In children over four years who had not been previously immunized it was thought wise to go through certain safety procedures, as in diphtheria there was an increasing reaction and sensitivity to diphtheria protein itself, which increased with age, and if there was any antibody at all present to diphtheria. The person was Schick-tested and Maloney-tested with 0·1 millilitre of a 1:100 dilution of pure diphtheria toxoid. If the result of the Schick test was positive and the result of the Maloney test negative, then immunization was started with 0·1 millilitre of undiluted alum-precipitated toxoid given subcutaneously, followed by 0·3 millilitre one week later and by 0·5 millilitre one month later. If the result of the Maloney test was positive, care must be taken and the dose reduced to one-tenth of what would be otherwise used (namely, 0·1 millilitre of 1:10 dilution) or the person even left unimmunized. For the recommended routine booster doses after the fourth year it would be safer to use 0·1 millilitre of the toxoid given subcutaneously every four years up to the age of twelve years. After that age reaction troubles were common, and sensitivity should be tested for. The Schick test itself was known to act as a booster shot; so that if there was sensitivity and a Schick test had been carried out, that might have produced as good a reaction as a dose of toxoid.

They were therefore faced now with a group of susceptible adults whose resistance had to be kept up by repeated boosters of some sort. That had been illustrated by some figures of the British Air Force during the war, when after five years only 66% were still Schick-negative.

Dr. McNair Scott said that the problem had received considerable attention recently because diphtheria had suddenly been found to be a major problem in the occupation troops of the United States in Germany after the war, when 2000 cases of diphtheria were experienced. Various investigators had demonstrated that the incidence of severe reaction was dependent on several factors—the age, the height of antibody level in circulatory blood and the dose of toxoid. The standard toxoids used in the United States had 40 Lf. units per millilitre, and therefore the recommended dose was 4·0 Lf. units—a dose of 0·1 millilitre—given to high school students. The reduction of the dose to 1·0 Lf.—in other words, a quarter of the routine dose—reduced the incidence of reaction still further and yet seemed to give a good booster effect. With that dose less than 5% of Air Force recruits had systemic reactions. Such a dose could probably be used for primary immunization of non-immune adults if two doses were given two weeks apart and a booster six weeks later. A preparation had been developed for experimental use, which combined 1·0 Lf. dose of fluid diphtheria toxoid with 5·0 Lf. doses of fluid tetanus toxoid in 0·5 millilitre amounts, and the preparation had been used in Massachusetts with good results on the antibody level. That was a problem which public health authorities needed to consider seriously.

Regarding tetanus immunization, Dr. McNair Scott showed a slide which summarized the routine procedure in the United States of America. After the fourth year, if triple antigen was not being used, 0·5 millilitre of an alum-precipitated toxoid was given, followed by a second dose one to two months later, and a third dose six to ten months later. A booster of 0·5 millilitre every three to four years was given, or on injury if no booster had been given within a year. A dose of 0·5 millilitre of a fluid toxoid was recommended on injury, because it had been shown by Miller that the antibody recall with fluid toxoid was faster than with the alum-precipitated toxoid. Very few reactions occurred, at the most a sore arm, and there seemed to be no excuse for not "pushing" widespread immunization against tetanus, particularly since only about 50% of cases of

tetanus arose from an overt focus. In about 50% of cases there was no history of wounding, and one therefore often could not even give the patients passive immunization. It had recently been shown that the active immunization against tetanus produced an immunity that lasted from eight to ten years. Therefore it would seem reasonable that the combined preparation of 1·0 Lf. unit of diphtheria toxoid and 5·0 Lf. units of tetanus toxoid given to adults every five to ten years would result in a well-immunized population against both diphtheria and tetanus. A slide then showed briefly the effect on the army of tetanus immunization. When the percentage of people without measurable antitoxin was noted, it was seen that women were entirely without antitoxin, whereas in men during their service period the percentage was very low.

In the virus and rickettsial fields the use of killed agents had a real although limited value among human subjects, and brief mention only was made of influenza virus vaccines, typhus vaccine and a number of others from the egg, and rabies phenolized spinal cord vaccine. Influenza vaccine had been very effective during the war, but suddenly in 1947 it ceased to be active and effective because the influenza virus had produced a strain with a different antigenic pattern, so that the old vaccine was useless. However, through the World Health Organization and international cooperation there was a free exchange of information about the various strains that were isolated from epidemics of influenza all over the world, and those strains were therefore known and could be and were incorporated into the current vaccines. So that actually they should be quite effective again and had been so in some small epidemics.

The use of Salk vaccine as a killed vaccine against poliomyelitis was then discussed. The vaccine was prepared by growing all three types of poliomyelitis virus in monkey-kidney tissue culture. After appropriate growth had taken place, each type was treated with formalin to a final concentration of 1:4000. The decrease in the virus activity was followed in the laboratory. When no more virus was detected, the process was allowed to go on for a time equivalent to twice the time taken for the virus to disappear. A slide was shown from Dr. Salk's report, and it was pointed out that in the laboratory it was possible to follow the decrease of the virus in what appeared to be a straight line. Dr. Salk extrapolated that straight line for another equal period of time, so that he anticipated that at the end of that period of exposure to formalin there would be one virus particle only in 500,000,000 millilitres. However, the inactivation seemed actually to follow a curve, and not the straight line of the extrapolation; for when the vaccines for the large field trial were prepared and tested very carefully for infectivity, quite a high percentage, perhaps up to 20% of batches, failed to pass the safety test. If it was remembered that one millilitre of each litre was tested, then it could be seen that there were in those that failed to pass the safety test one infectious particle in 1000 millilitres. When the vaccine prepared by one company was used in about 5,000,000 children, there were about 50 cases of paralysis which seemed to be due to the vaccine; that put the incidence at one in 100,000. That made the problem of killing the virus a difficult one. Also with regard to antigenicity, one must have a very high titre virus, because antigenicity was also lost during the process of killing but at a slower rate. Various suggestions had been made to overcome those defects, such as the use of a longer time in formalin, or the introduction of a less virulent strain of poliomyelitis virus into the vaccine. They were both under investigation at the moment.

The vaccine as used had been a perfectly good antigen of all three types of virus, as was shown by illustrations from Dr. Salk's paper. From those slides it was demonstrated that, as Dr. Salk had suggested, the routine procedure should be two doses three to four weeks apart, and then a booster dose not less than seven months later, perhaps ten months later. In a group of people who had had no antibody before they were vaccinated, at the end of ten months after their primary inoculation many still had antibodies, although some had lost measurable antibodies. However, when they were given a booster the level of antibody rose to very great heights, similar to that reached after natural infection. The rise occurred within three to four days. Some children after primary vaccination acquired a natural infection, and their antibody level rose to heights similar to those of children having booster injections. That was an important observation, because it would presumably be partly through the exposure of those partially immunized persons to natural infection that they would manage to keep their antibody level up for a long period of time. That was not necessarily the case, because investigation had suggested in the past that poliomyelitis antibodies did remain in the blood-stream for a long period of time without exposure to subsequent infection.

The Salk vaccine had been a great step forward in the understanding of poliomyelitis and the antibody response; how applicable that sort of vaccine would be to mass immunization only time would tell.

Dr. McNair Scott then briefly considered the matter of living antigens. He said that it might be that they would eventually prove a more promising approach to the goal in a number of instances. At present vaccinia and yellow fever vaccines were well known and effective agents of that sort. In the United States the multiple pressure method was used for smallpox vaccination, and it was suggested that primary vaccination be carried out at about six months of age, at the end of the routine immunizations with the triple vaccine, and repeated at three-year to five-year intervals throughout life and on exposure. That was the ideal. Vaccination was not compulsory in the United States, but going to school was, and nobody could get into school without vaccination. Yellow fever vaccine was a living vaccine, and that was given only to a population exposed to special risk. Those two living virus vaccines were avirulent mutants of virulent viruses. Vaccinia vaccine was a mutant of smallpox vaccine and yellow fever an avirulent mutant found in the laboratory after passage through eggs and mice. It must be considered therefore that the poliomyelitis vaccine problem might be approached in that way, and at present at least two groups in the United States were working on the matter. Both had shown the development of antibodies in human volunteers when fed with living but avirulent viruses—meaning avirulent as far as all laboratory animals were concerned. It might possibly be that, as with the early yellow fever immunization, the giving of a living vaccine might be covered, as it were, by temporary passive immunization by γ globulin.

One other living vaccine had been used very widely in many parts of the world including the United States. That was B.C.G. vaccine against tuberculosis. The beautiful statistical work of Aronsen had opened up the value of that product, and clinical experience throughout many parts of Europe and Asia had shown its effectiveness in decreasing the incidence of tuberculosis. In Philadelphia it was used only in groups at special risk, such as contacts and newborn Negro infants, as tuberculosis was more serious in the Negro race, and it was also offered to nurses and medical students who were Mantoux negative.

In summarizing, Dr. McNair Scott said that it seemed clear that certain plagues could be controlled by immunization procedures at present available, including diphtheria, yellow fever, smallpox and the non-epidemic but highly lethal infection of tetanus. The control of those was available, but the actual implementation of the control needed a great deal more work. The plague of poliomyelitis was not yet conquered, but big steps had been taken. Immunization could be further provided for those at special risk, such as against pertussis for the very young infant, and B.C.G. vaccine for certain infants and other individuals especially exposed to tuberculosis. Finally, it seemed useful once in a while to review the theoretical bases for immunization procedures and what was really being aimed at in immunization programmes.

PROFESSOR S. RUBBO opened the discussion and commented on the extrapolation of the graph from Dr. Salk's paper showing the killing of virus particles in poliomyelitis vaccine. He said that that graph was plotted on a logarithmic scale; and if it followed the usual unimolecular reaction of disinfection, then a straight line would be expected. However, the facts suggested it did not follow the straight line. He thought that the latest view on inactivation of Salk virus was to extend that period to three times the actual length of time observed to inactivate the particles as determined by tissue culture. Professor Rubbo then asked whether any investigations on the intradermal method of immunization injections had been carried out. It had been implied that the Schick test acted as a booster dose. That might be a way of saving pinpricks with such immunization procedures as those against diphtheria, tetanus and pertussis. It was, he understood, used as the routine for "T.A.B." in Malaya.

Dr. McNair Scott, in reply, said that he did not know of investigations into the use of intradermal injections for immunizations. He thought that they would be more painful and more difficult to perform in infants than the routine 0·5 millilitre subcutaneous injections. He said that in the armed forces during the war he thought that the United States Navy did their "T.A.B." immunizations by intradermal

methods and the Army the other way. There was no doubt that a rise in "T.A.B." antibodies was obtained by the intradermal route.

DR. S. WILLIAMS said that the lecture had been extremely helpful, had confirmed many things and had given a warning for which preparation must be made. It had shown clearly the pitfalls one ran into in an efficient immunization campaign. He said that the local immunization campaign was good but had not quite wiped out diphtheria, and the problem pointed out of increased incidence in the older age group was new in Victoria. He asked two questions: firstly what was the current view in the United States about the "McCloskey phenomenon" (in other words, paralysis associated with immunization injections), and secondly, had a formalin-killed vaccine been tried in yellow fever.

Dr. McNair Scott said, in reply, that the "McCloskey phenomenon" had been confirmed in the United States, and good studies in New York State indicated that routine immunization, particularly for diphtheria and pertussis, did act as provoking stimuli for poliomyelitis. The limb inoculated was the limb that was paralysed, and there was an increase of paralysis in the inoculated group. However, it was not a very great increase. When McCloskey had first reported it, and it was confirmed in England, the recommendation was that no routine immunization should be given to children over six months during the summer poliomyelitis period. However, he thought that that had not been good advice, as the result was to prevent a lot of children getting their routine immunization, and it had upset the public. The present advice, and probably the wiser advice, was that there should be no alteration in routine immunization except during an epidemic of poliomyelitis in the environment.

He did not know of any attempt to use formalin-killed yellow fever vaccine. Live vaccine was so effective that it had almost wiped out yellow fever where it had been used.

DR. D. HAGGER asked whether the advice was not to immunize children aged under six months during an epidemic of poliomyelitis.

Dr. McNair Scott said that it had been suggested that the child under six months was relatively insusceptible to poliomyelitis, but he was not sure that that was true. Although the incidence of poliomyelitis might be a little less under six months of age, the mortality was many times greater. However, advice given was actually to go ahead with the primary immunization in the six-months-old baby, but no booster shots were given to children in older age groups in epidemic conditions.

DR. H. WILLIAMS asked why, in Philadelphia, mass immunization of children with B.C.G. was not carried out. In Victoria it did not seem necessary from the very low incidence of tuberculous infection in children, and he wondered whether conditions in Philadelphia were similar. In Victoria only 1·5% of approximately 10,000 children reacted to tuberculin under the age of five years. Between five and fifteen years 3·5% reacted. In children aged ten to fifteen years 5% reacted. The mortality in 1952, the latest figure available, was 0·25% of all deaths under the age of fifteen years; so the incidence of the disease was extremely small.

Dr. McNair Scott said that he could not quote figures, but the problem was not a great one except in those at special risk. B.C.G. was not entirely without certain discomfort, in that one always got an ulcer.

DR. J. FORBES asked whether any of the virus manufactured in the Salk laboratory had been incriminated as containing live virus, or whether Dr. McNair Scott had referred only to the virus from one other laboratory.

Dr. McNair Scott said that he did not think any other laboratory was incriminated. The Cutter laboratory was first in the field, and the vaccine was used for the first 5,000,000 children in the mass immunization. That laboratory had done everything that they were asked to do in terms of safety precautions. None of the other companies had made available large amounts of vaccine, and all were stopped before they had vaccinated a large number.

DR. M. POWELL said that he would like to see the triple antigen made the standard product for use in Victoria, and thought that more publicity should be given to it by the makers. He wished for Dr. McNair Scott's comments on the use of triple antigen as against the other single antigens.

Dr. McNair Scott said that it had become almost standard procedure to use triple antigen, but only recently. However, it was necessary to have available vaccine that did not contain one or other antigen, particularly pertussis, because if there was a severe reaction with triple vaccine, the first thing to drop out was pertussis. However, the triple vaccine was an excellent antigen.

Dr. M. Powell also said that he did not think that the importance of tetanus immunization had been brought home fully to the general mass of medical people in Australia until recently.

Dr. McNair Scott then said that he would be interested to know the incidence of tetanus in such a highly agricultural country as Australia.

DR. FORBES MACKENZIE offered the information that there were about 30 cases of tetanus per year notified in Victoria, and about half of those affected died.

DR. V. COLLINS asked what would the American procedure be for unimmunized patients who had skin sensitivity to antitetanic serum. It had been suggested that if they were sensitive then the antitoxin would be excreted rapidly, and therefore it was not worth immunizing them, but they should be given a dose of toxoid at that time.

Dr. McNair Scott said that he thought they would follow the usual desensitization procedure and give antitoxin, but also give toxoid in another site at the same time. Antitoxin might be excreted fast, but probably there would be sufficient to keep the toxin under control. Penicillin would be given also to have a bactericidal purpose.

DR. D. HAGGER asked for some further information about encephalitis as a complication of pertussis vaccination, inquiring what was its frequency and whether the pertussis vaccination was actually the cause. He said that reactions to vaccination were reasonably common, however, and it would mean stopping vaccination in a considerable number of cases if that was regarded as a contraindication.

Dr. McNair Scott said that he did not mean to indicate that one did not get febrile reactions. One did. However, the child who got a temperature of 105° F. or an exceptional reaction might raise the question of advisability of giving pertussis in the next injection. If the child had a convulsion, he thought very strongly that pertussis vaccination should be ceased. He could not give an answer to the question of incidence, however. The development of encephalitis was a widespread experience, but not a very common one; however, everyone should be aware of the position and beware of the relationship. There was some experimental work in mice showing that pertussis vaccine made them more sensitive to histamine, but whether that had any relation to the problem he did not know.

DR. R. WALL asked whether the vaccine should not be used for any child who had had a previous convulsive episode unrelated to the giving of vaccine.

Dr. McNair Scott said that this was a difficult problem. Some people would go as far as that; and if one wanted to be on the safe side, it would perhaps be the thing to do. However, it was necessary to balance the good and the ill. One could get encephalitis from pertussis itself.

DR. J. COLEBATCH asked whether Dr. McNair Scott's statement that immunization against pertussis in those over four years was of debatable value was based on the fact that reactions occurred, or on the possibility of the person's being a carrier and bringing it home to children in the house. He also asked if there was any place for immunization against pertussis before the age of two to three months, as that was the time babies were often taken to health centres amongst other children in Australia, and were likely to contract pertussis in severe form. Lastly, he asked whether in a child who suffered from convulsions following pertussis immunization, there was any place for continued immunization with lower dosage and a greater number of injections, or whether such a child was sufficiently immunized already against pertussis.

In answer to the first question, Dr. McNair Scott said that if there were young children in the family and one wanted to prevent the school child from bringing pertussis home, it would be best to continue the immunization through school age. If there were no younger children, then it might be best to let the child get a mild attack of pertussis and acquire a more permanent immunity. Also pertussis vaccine was an increasing cause of reactions.

In answer to the second question, he thought that if environmental and social problems suggested that immunizing early might be important, then there seemed no contraindication to that. A high antibody level would probably not be obtained, but there was no subsequent lack of booster effect.

In answer to the third question, he thought that the child would not be fully immunized, and a decision would have to

be made on the relative importance of continuing immunization or suffering brain damage.

DR. E. NORTH asked whether in the triple antigen it was the custom to use adsorbed toxoid. In Australia fluid toxoid was used, largely because of the close connexion between mixed antigens and poliomyelitis.

Dr. McNair Scott said that it was almost routine to use the adsorbed antigens. They were better antigens and longer lasting, and it was thought that those advantages offset the danger of poliomyelitis.

DR. H. SINN asked whether Dr. McNair Scott would agree that it was best not to carry out pertussis immunization if the child had a respiratory infection of any sort at the time, and whether, if the injection was delayed until the infection had subsided, there would be any difference in the development of immunity reaction.

Dr. McNair Scott said that he would agree with Dr. Sinn in his suggestion; he did not think it would affect the ultimate degree of immunization.

DR. S. FISHER, in referring to pertussis vaccine, said that there was experimental evidence that the encephalitis might be due to the activation of some latent virus infection. He thought that the figure stated for incidence of encephalitis was one in 100,000 of the population. Dr. Fisher also said that the efficacy of triple antigen had not yet been accepted by all workers, and at present the Commonwealth Serum Laboratories were producing the other antigens and giving practitioners the benefit of making a choice.

Dr. McNair Scott said that he did not know the figure for incidence of encephalitis.

DAME J. MACNAMARA asked Dr. McNair Scott if he would elaborate his remarks regarding the use of hyperimmune serum for pertussis. She thought it would be of advantage in young babies because of the greater danger of permanent paralysis as the result of the "McCloskey phenomenon".

Dr. McNair Scott said that antipertussis hyperimmune serum was actually made at the Children's Hospital in Philadelphia. It was valuable both as a prophylactic measure and in treatment of early cases, but it was extremely expensive to prepare and would never replace active immunization.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THERAPEUTIC VALUE OF EUCALYPTUS.

To the Editor of the *New South Wales Medical Gazette.*

Sir,

While Mrs Gimbert and de Valcourt, as it appears in your last number are squabbling in France about the priority of their discovery of the value (medicinally) of the products of the Eucalyptus, you will, I doubt not, allow me to use your periodical to state that, shortly after my arrival in this colony, in the beginning of 1838, I went to practise in a country district, and had, consequently, to travel through forests of these trees. In doing so, I could not fail to observe the "tears" of gum exuding from rifts in the bark, which had so much the appearance of the Kino of commerce, that I was induced to taste. The astringency of the product, so much resembled that of the Kino, that I, not then having any extensive repertoire of medicines, was tempted to try its effect in cases of diarrhœa. On doing so (using it pulverized either alone, or in combination with the common chalk mixture) I was very thoroughly satisfied of its efficacy. In fact I found it quite as good in its effects as the imported Kino or catechu of commerce if not more so.

I afterwards found that "Botany Bay Kino" (from Thomson's Materia Medica) had been imported into England as early as (I think) 1823 or 1826 but the trade seems to have been afterwards discontinued nor do I remember having seen it recorded among our articles of export, although Neligan states that "Botany Bay Kino is sometimes met with".

For myself, I have, from my experience, a high opinion of it as a powerful astringent, and would advise medical practitioners residing in country districts to use it. It may be collected without much trouble in any quantity, and, I imagine, that by tapping the trees when the sap is rising, it might be obtained in sufficient quantity to supersede the imported articles.

Here is another hint for the encouragement of "native industry" without the necessity of a "protective tariff".

I am, Sir, &c.,

ISAAC AARON.

Sydney,
December 27, 1871.

Correspondence.

HOSPITAL POLICY.

SIR: There have been discussions recently on a hospital policy for the British Medical Association, but it does not appear to me to have been sufficiently stressed that there is a great difference between country and city hospitals, and a policy which is satisfactory for the city will not suit the country hospitals. In the city, only a select few are on the honorary staff of the big hospitals. To be appointed to the staff of one of these hospitals confers a distinction on the doctor, which compensates him to some extent for treating patients free of charge, but his treatment is more in the nature of an advisory capacity. He has residents at his disposal to carry out routine work. He may have to do difficult operations, but he has not to come back later and give the patient a blood transfusion or other post-operative treatment. All that is done by subordinates. He has not got to treat his well-to-do patients in the public wards, as there are private hospitals available to him.

How differently placed is the country practitioner! All doctors are automatically on the staff of the hospital in the town in which they practise; so no distinction is conferred by being an honorary doctor to the hospital. In a large country hospital there may be one resident where they could do with five. Mostly there are no residents. There are no private hospitals and only few private beds attached to the country hospitals. So the unfortunate country doctor is obliged to send his patients into the public ward, perform a difficult operation and then do the after-treatment. And all for nothing, even though the patient has ample means and pays into a benefit fund. This state of affairs is ridiculous and absurd.

In regard to obstetrics, the difference between city and country is even more marked. Only a few private beds are set aside in maternity blocks, and the accommodation is much the same for private and public; so many women whose husbands earn £20 a week or more elect to be public ward patients. The country doctor with no residents to do the routine management may sweat for hours on a case, losing a night's sleep or being called away from a busy surgery. And all honorary! The city obstetrician has his difficult cases also, but once he puts things right he can leave; but not the country man, who has to see the case through to the end.

So a common policy for city and country is impossible; and while an honorary system may be practical for the city, it is outmoded in the country and should not be tolerated any longer.

Yours, etc.,

E. B. FITZPATRICK.

Tamworth,
New South Wales,
Undated.

LUMPS IN THE BREAST.

SIR: During the recent congress meeting in Sydney a paper was read in the Section of Surgery by Mr. A. C. Thomas on "Lumps in the Breast" (as reported in your journal of October 26, 1955). In opening the discussion on that paper I suggested that many difficulties in managing these patients arose because one is tempted to give a "name diagnosis" to a pathological condition on clinical evidence which can be inadequate and often misleading.

In reference to this the remarks which I made were along these lines. When a patient comes complaining of a lump

in the breast, it is very difficult after a careful clinical examination to look wise and say "you have a lump in the breast". To satisfy the patients' desire for a name diagnosis and to save ourselves the embarrassment of not being able to add anything to their diagnosis, an attempt is made to classify it pathologically (and act accordingly) on insufficient evidence.

To me it seems a pity that we as a profession do not have some impressively sounding name which means nothing more than a "lump in the breast". Perhaps we could coopt some of our dermatological friends and easily solve this problem. To quote an example we could call it a "mastoma". It could be taught to students and used as a name diagnosis to satisfy both patient and doctor. When the name is given and the patient makes the inevitable inquiry of "What's that, doctor?" one could look them squarely in the eye and say: "It is a condition we often see in the breast, its cause is various, but its treatment clear cut—it should be removed without further delay."

There should be universal recognition of the fact that failure to classify these lumps on clinical evidence does not mean that one's clinical acumen is failing or one's diagnostic ability becoming defunct. A name for a lump in the breast such as "mastoma" would, as I have said, satisfy the patients in providing a name diagnosis, and satisfy the clinician, for having made this diagnosis the treatment is straightforward—biopsy to prove the pathological nature of the lesion and appropriate treatment forthwith.

If some such plan were adopted, the management of lumps in the breast confused at present by one's inability to "give it a name" would then be well defined. Also the percentage of carcinomas diagnosed, while still within the limits of a Stage I classification, would increase.

Perhaps there are some readers who may find something useful arising out of these suggestions, and I submit them accordingly.

Yours, etc.,

217 Macquarie Street, Sydney,
November 8, 1955.

R. P. MELVILLE.

THE MEDICAL SERVICES OF THE AUSTRALIAN ARMY.

SIR: I have been most gratified to see that the problem of enlisting medical officers for the Australian Regular Army is being approached in a more realistic fashion. However, I think that before unqualified approval is given to the scheme whereby undergraduates are financed through the later years of their course in return for a number of years' service, a more critical appreciation of the disadvantages of the service at the moment should be made.

As one of the earliest enlistees in the regular Royal Australian Army Medical Corps (and one of the earliest resignees), I found that most young doctors associated with the service were not only satisfied with the pay, but that at least two were attracted by its generosity. Dissatisfaction was levelled rather at the conditions of work.

A medical officer enlisting in those days was expected to fill one of the numerous administrative vacancies, and presumably to deal with the pure medical problems in his spare time. For a conscientious officer to take on these responsibilities, without training, under an accounting system which has been described by Field Marshal Viscount Montgomery as designed under the assumption that every officer is a potential criminal, can be a heart-breaking and soul-destroying job.

The camp hospitals were relatively well equipped in everything except clinical interest. This was due not only to the limited source of material, but even more to a policy whereby there was definite discouragement of any case more serious than "U.R.T.I." or diarrhœa being kept and not immediately evacuated to the Repatriation General Hospital.

The camps themselves are situated in the country, which, though healthy, is a limiting factor in keeping in touch with medical thought in the cities (Puckapunyal is 66 miles from Melbourne), and this is a most definite contradiction to post-graduate study.

It is to these conditions that graduates with minimal medical experience will be posted for four years. During this time they will have treated no women or child patients, only young males who are probably slightly more fit than the corresponding general populace. On completion of their contract they will have no choice but to have at least one year's medical reeducation, or to stay in the army because they are unsuited and untrained for anything else. As a large proportion of them will have joined the service only to resolve financial difficulties in their undergraduate days, this seems a heavy price to pay.

Most people will agree that the worth of the recent graduate for further training is first apparent during his residence; so potentially skilled doctors will be denied post-graduate training as senior residents at the time when such training would be most beneficial to them. Conversely the Royal Australian Army Medical Corps would be boosted in quantity, but neither in quality nor prestige.

As the service apparently must be made more attractive, it seems to me that it is more logical to deal with the conditions of service, rather than the conditions of entry. Worthwhile candidates will be attracted by the prospect of an interesting and useful professional life rather than by a cheap medical education.

It is proposed that as an alternative the term of enlistment be six years, open to graduates only; so that the officer could be seconded for civilian training in any aspect of medicine that he chooses for a full two years (which is the generally accepted period for diploma training) at an approved hospital, preferably a teaching hospital. If this two-year period were made available after the second year of service, the army would gain the additional advantage of having young specialists for the final two years of their enlistment; but they must be given the opportunity of practising their specialty, in addition to general medical duties if necessary, and not forced to rot in unwanted and uninteresting administrative postings. It is felt that the presence of these specialists would in turn raise the prestige of the corps immeasurably more than the sheer weight of numbers of poorly trained general duties personnel.

If there are any who really prefer administrative work, this two-year training period could be spent in an attachment to a British army headquarters, where experience could be gained with real troops on the ground or at an army training establishment such as the Staff College. Then the future controllers of our martial destinies will have been taught their duties properly, and not had to pick their information up as best they could.

Finally, to increase the interest of the type of practice, could not a service be extended to army dependants? Such a service is given by the Royal Army Medical Corps, and was given many years before the *National Health Act* was introduced in Britain, such a service was given by area medical officers before World War II in Australia, and such a service was given in the British Commonwealth Occupation Forces. In an army of the size of the Australian Regular Army the financial competition to civilian practitioners would be negligible.

Yours, etc.,

24 Cliff Street,
Watson's Bay,
New South Wales.
November 11, 1955.

E. MANCHESTER.

AN ADDRESS.

SIR: The address by Dr. A. E. Lee which appeared in the journal of November 5 last deserves close attention by us all. In admirable fashion he has succinctly covered developments in medical practice in the post-war years which have affected in various ways each and every one of us.

If he has indulged in some plain speaking, that, too, is to be commended because errors and abuses can be corrected more easily when brought out into the open. However, this privilege of plain speaking belongs equally to all members of our Association, and maybe a little of it regarding our Federal and State Councils will not be out of place at this juncture.

Both the Federal and Queensland State Councils have been making commendable efforts to stabilize fees at a reasonable level—particularly to protect medical insurance funds. Members of both councils regard the fees at present charged by the majority of members as being fair and equitable to both doctor and patient. Why is it then that, whenever negotiations take place with official and semi-official bodies, a lower fee than the ruling fee is agreed upon by our repre-

sentatives? Whether it is a repatriation fee, a workers' compensation fee, a local authority fee or any other fee, it is always lower. It has been stated that it is because the payment is certain, but would the usual fee be less certain? And surely the mass of paper work entailed merits a greater rather than a lesser emolument.

Decisions are often taken by our councils without prior approval of members, and agreements are presented as *faits accomplis*. For example, the last meeting of Federal Council adopted as its official policy a memorandum drawn up by Dr. H. C. Colville. Nowhere in the notes of the meeting published in the journal of September 24 is reference made to any prior submission to State councils.

To bring our central councils more in touch with the periphery of the profession some radical alteration is overdue in their composition, and machinery must be devised to give the John Does of the profession a real voice in the ratification of important agreements before they are implemented.

Yours, etc.,

Bowen, Queensland, November 12, 1955.

PETER R. DELAMOTHE.

PENICILLIN AND FURUNCULOSIS.

SIR: I would like once again to record my opinion that it is useless to try to cure furunculosis with penicillin. Of the hundreds of cases I have seen in recent years, the large majority are relapsed penicillin-treated. Of the latest two, within the last week, one was treated by a general practitioner for a "boil" with penicillin, and four more "boils" promptly appeared; the other, treated by a dermatologist with penicillin for a "boil", developed three more immediately. Yet these two doctors persisted in a form of treatment which was obviously useless, opposed to the patients' interests and possibly tragic, since these people might have developed a staphylococcal septicæmia, and penicillin, as a life-saving agent, could have been worthless.

Maybe I am seeing only the unsuccessful relapsed penicillin-treated cases and not the possibly successful ones. But, on the basis of a very long and wide experience, I am beginning to believe that such a thing as a penicillin-sensitive strain of *Staphylococcus aureus* (*pyogenes*) is ceasing to exist, and we are helping the process.

In Queensland, particularly in the summer and, curiously enough, almost exclusively in males, furunculosis is a very serious industrial and medical problem, which needs rational thought instead of blind stabs of a valuable substance which may save life but will not cure boils.

Yours, etc.,

Ballow Chambers, Wickham Terrace, Brisbane. November 16, 1955.

J. V. DUHIG.

APPOINTMENT OF INTERN WARDEN BY THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

SIR: The announcement by the Post-Graduate Committee in Medicine in the University of Sydney of the appointment of Dr. J. Colvin Storey as intern warden will be welcomed as a sign that the University is interesting itself in the education and training of young graduates during their period of residence in hospital.

The sudden termination of all university influence in medical teaching which occurs on graduation has for some time been a source of deep concern to experienced clinicians and others. In all too many instances the young graduate's period of residency is too brief and too haphazard; and whilst it is most essential to encourage initiative and originality of thought, it is nevertheless desirable to continue some further planned and supervised training. At present the necessary organization to ensure this exists only in rudimentary form—or not at all. It may be necessary in some cases to arrange for representatives of the University to place before hospital boards their plans for this training, and to carry them out on a basis of close cooperation.

All this is very necessary, and should be considered an integral and important part of medical education. It is to be hoped that the example set by the University of Sydney will be followed in other universities with medical schools. Adequate training of resident medical officers is of fundamental importance, and the hospital is in an excellent position to play a part in this work, an activity which, it should be remembered, advances the standard of its service to the community.

Yours, etc.,

Brisbane, November 22, 1955.

E. S. MEYERS.

REDUCTION OF INTUSSUSCEPTION BY BARIUM ENEMA.

SIR: Anent the recent article on the reduction of intussusception by barium enema, the following incident is of interest. Years ago the late Dr. E. D. Edwards, of Innisfail, desired an opaque enema examination of an infant in its first year acutely ill with abdominal distress. It was during the wet season, and the sixty-mile road, not bituminized at that time, was untrafficable. Being too ill to travel the distance by rail, a plane was chartered and the infant duly arrived in Cairns. An opaque enema was administered without screening, after which the infant had recovered from the acute distress. The weather was too stormy for the immediate return of the plane and the patient was returned to Innisfail by train. A telephone message from Dr. Edwards started with: "What on earth did you do to the child?" He was so amazed at the sudden and complete relief that he requested me to send several doses for administration if the symptoms recurred.

Yours, etc.,

Cairns, North Queensland, November 17, 1955.

H. FLECKER.

THE PLENARY SESSION ON CANCER AT THE RECENT CONGRESS.

SIR: I am glad you have published a letter from Dr. Belisario with reference to his contribution to the discussion at the plenary session on cancer during the recent Congress. I can bear witness to the accuracy of his remarks, both as regards his own question and the reply by Sir Stanford Cade. Sir Stanford's observations, both in tone and content, were unworthy of a man of his stature, and whilst a few were entertained thereby, a great many were irritated. If guest lecturers adopt the "when I speak, let no dog bark" attitude, then the value of these sessions will be lost. An intelligent contribution by any medical man is surely entitled to a courteous answer by a lecturer, after at least making sure he understands the question. When a question is put by an acknowledged leader in his field, he should be assured that he will not be belittled.

Yours, etc.,

Bulli, New South Wales, November 20, 1955.

BERTRAND A. COOK.

MODERN PSYCHIATRY AND ITS RELATIONS TO MEDICINE AND SURGERY.

SIR: I read with interest the essay by Dr. H. B. Ruddock and must congratulate him on dealing well with a problem child such as "Modern Psychiatry and its Relations to Medicine and Surgery". It is easy to find fault with the details of such a wide subject; but because I feel that some of them are of fundamental importance I wish to make these points:

"The qualities of a nerve impulse leaving a neuron depend on the nature of the impulse before entering it, together with whatever alteration has taken place while it is passing through the cell." This statement of Dr. Ruddock's is in direct opposition to the "all or nothing" function which is attributed by physiologists as a quality of the nerve impulse. So logically Dr. Ruddock's postulate of "impressed neurons", based on a false assumption, is itself false.

However, when the integrative activity of the nervous system is studied large numbers of neurons are involved in reflex arcs. Eccles has shown that use or disuse can markedly change the reflex response of a given stimulus. The change probably occurs in the terminal dendrites in the region of the synapses.

If Dr. Ruddock's argument is transposed and the phrase "impressed neurons" is replaced by "impressed pathways", it would then be based on sound physiological tenets.

I question another statement of Dr. Ruddock: "The mind can receive impressions from the outside world only by the sense organs. . . ." Here I quote from the Seventh Arthur Stanley Eddington Memorial Lecture by H. H. Price, Professor of Logic in the University of Oxford: "It is an indisputable fact that supernormal cognition does occur." This criticism is not directed at Dr. Ruddock's method of argument as such, but at the "materialistic conception of human personality" which follows from this basic assumption.

Yours, etc.,

ROBERT COLLIN.

Physiology Department,
University of Western Australia,
Perth.
November 21, 1955.

Reference.

ECCLES, J. C. (1953), "Neurophysiological Basis of Mind", Clarendon, Oxford.

PRICE, H. H. (1953), "Some Aspects of the Conflict between Science and Religion", Cambridge University Press.

MEMBERSHIP OF THE BRITISH MEDICAL ASSOCIATION.

SIR: About a week ago the writer received a note from the Medical Secretary of the British Medical Association, in which Dr. J. G. Hunter stated that he had received advice from the London Office to the effect that I shall have completed fifty years of membership at December 31, 1955. Referring to a "Scrap Book" in which for many years I have pasted things which I thought would be of interest to myself in later years, I find there that I had kept a notice *re* a meeting of the British Medical Association (then held at the Royal Society's Rooms in Elizabeth Street, Sydney). The date of the meeting was Friday, April 27, 1905, when I was a resident medical officer at the Royal Prince Alfred Hospital. However, it is about my late brother, Dr. George Henry Walton Smith, that I wish to comment; he died in April last year at the age of eighty-five years, and he had then been a member of the British Medical Association for about sixty years. I am anxious to know whether the combined total (110 years) is a record for two members of the one family.

Yours, etc.,

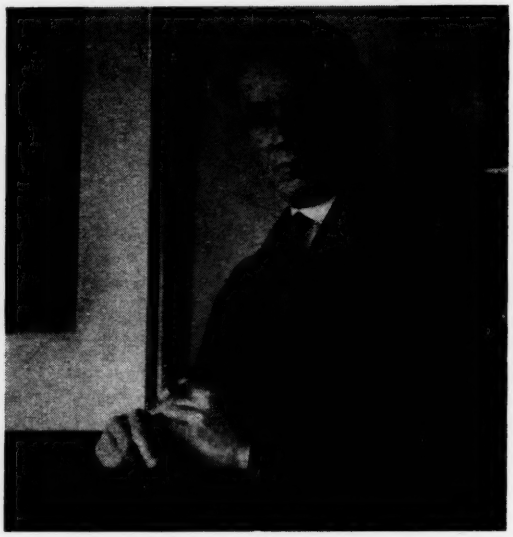
P. E. WALTON SMITH.

30 Bondi Road,
Bondi Junction,
New South Wales.
November 27, 1955.

Obituary.

GEORGE ARTHUR BROOKES.

GEORGE ARTHUR BROOKES was born at Brisbane on February 22, 1880. He was educated at Brisbane Grammar School, and entered the Queensland Public Service; but he had an overwhelming desire to become a doctor, and at the age of twenty-two years he entered the University of Sydney. He was not well off, and had a struggle to make ends meet; however, he shared the John Harris Scholarship in third year with E. A. Brearley and this was a great help. He was one of the four prosectors when S. A. Smith was demonstrator. He had a brilliant university career, and graduated in 1907. After residency at Royal Prince Alfred Hospital he set up in general practice at Petersham, New South Wales, and was later appointed to the staff of the Lewisham Hospital. During the first World War he served in France and England with the Australian Imperial Force. After demobilization he returned to his practice at Petersham, and married. In 1920 he went abroad for post-graduate study; he gained the F.R.C.S. (England) in 1921; then, attracted to ophthalmology, he worked at Moorfields Eye Hospital in London, and in Vienna, and gained the D.O.M.S. (London) in 1923. On his return to Australia he set up in Macquarie Street, and became honorary ophthalmologist at Royal South Sydney Hospital, St. George District Hospital and Lidcombe Hospital. During the second World War he "did his bit" by giving untiring service at an aid post in Surry Hills. In 1945 his wife died; he remarried in 1946. He carried on his practice until ill health forced him to retire in February, 1955; he died on August 16, 1955.



George had a brilliant mind and a wide culture. He was devoted to music, with Bach his favourite. He had a deep love for, and knowledge of, painting, Australian in particular; his "Crossroads", by Hilder, has been bequeathed to the National Art Gallery. He read widely and intelligently—afternoon teatime in the surgeons' room was invariably devoted to an enthusiastic exposition of the latest book he had read. He was a first-class ophthalmologist, and his eye surgery was a delight to watch. He was kind and generous, and tolerant of everything but stupidity, which his quick mind could not understand, and which moved him to swift impatience. His patients liked and respected him, and children loved him as much as he loved them.

Research.

OPHTHALMIC RESEARCH INSTITUTE OF AUSTRALIA.

ATTENTION is drawn to the existence of funds in the Ophthalmic Research Institute of Australia available for research in any branch of ophthalmology. Further particulars can be obtained from the Honorary Secretary, Dr. W. Deane-Butcher, 235 Macquarie Street, Sydney.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following announcement was published in the *Government Gazette of the State of New South Wales*, of November 18, 1955.

Notice is hereby given that the name of Dr. Frank Solomon Hansman (3955), 143 Macquarie Street, Sydney,

M.B., Ch.M., Syd., 1920, is expunged from the list of names removed from the Register of Medical Practitioners for New South Wales by order of the Board, dated 13th October, 1955, published in the Government Gazette, number 120, dated 21st October, 1955, he having paid the Annual Roll Fee within the prescribed time. The inclusion of his name therein was the result of a clerical error.

P. E. COSGRAVE, Secretary.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 60, of November 17, 1955.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

The Short Service Commissions granted to the following officers are extended until the dates shown: 2/40110 Major D. C. Cook, 26th October, 1955, and 3/40109 Captain (Temporary Major) A. O. Donald, 23rd December, 1956.

To be Temporary Major, 10th October, 1955.—3/40109 Captain A. O. Donald.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/39189 Major V. E. Sampson, M.C., is appointed from the Reserve of Officers, 30th September, 1955.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—The provisional appointment of 2/130111 Captain J. A. Jaconelli is terminated, 5th October, 1955. To be Temporary Major, 20th October, 1955: 2/101562 Captain J. V. Roche. To be Captain (provisionally), 6th October, 1955: 2/130111 Joseph Anthony Jaconelli.

Western Command.

Royal Australian Army Medical Corps (Medical).—5/26397 Captain (provisionally) W. I. Gordon ceases to be seconded, relinquishes the provisional rank of Captain, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Western Command) in the honorary rank of Captain, 18th October, 1955.

Tasmania Command.

Royal Australian Army Medical Corps (Medical).—The provisional ranks of the following officers are confirmed: Captains 6/15417 R. M. Hughes and 6/15388 R. J. Connolly.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

Northern Command.—To be Honorary Captains: Raglan Fitzroy Somerset, 7th October, 1955, and Francis Patrick Sullivan, 10th October, 1955.

Eastern Command.—To be Honorary Captain, 21st October, 1955: Graeme John Morgan.

The following officer is retired, 12th October, 1955:

Western Command.—Captain H. N. Guthrie.

The following officers are placed upon the Retired List (Western Command) with permission to retain their ranks and wear the prescribed uniform, 12th October, 1955:

Western Command. — Lieutenant-Colonel (Honorary Colonel) L. E. Le Souef, O.B.E., E.D., Majors S. G. Taylor and F. H. Vincent, Captains S. Finklestein and R. S. W. Thomas.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

Flight Lieutenant M. A. May (013705) is granted the acting rank of Squadron Leader, 4th August, 1955.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED NOVEMBER 19, 1955.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	3(1)	1	4	..	..	..	..	..	8
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	1(1)	..	..	..	..	..	..	1
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	1(1)	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	7(5)	8(7)	5(5)	..	..	..	2	..	22
Diphtheria	..	2(1)	1	..	3(3)	..	..	..	6
Dysentery (Bacillary)	..	..	4(3)	..	1(1)	..	..	..	5
Encephalitis	..	..	..	1(1)	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	56(17)	60(33)	..	20(10)	1(1)	..	..	..	137
Lead Poisoning	..	..	1	1(1)	..	..	..	..	2
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	13	..	..	..	..	..	13
Malaria	..	..	1	..	1	..	..	..	2
Meningococcal Infection	1(1)	2(1)	..	..	..	..	..	..	3
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	1(1)	..	..	1(1)	..	..	..	2
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	3(1)	..	6(2)	1(1)	..	..	..	10
Puerperal Fever	1	..	1(1)	..	..	..	..	..	2
Rubella	..	135(115)	..	2(1)	..	..	..	..	137
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	11(6)	14(8)	18(10)	7(7)	..	..	..	..	50
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	..	..	..	..	1
Trachoma	..	..	..	..	26(6)	..	..	..	26
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	52(38)	14(10)	2(1)	5(4)	12(5)	5(1)	3	..	93
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

The short-service commission of the following Flight Lieutenants is extended to the date indicated: J. R. Harrison (013663 (acting Squadron Leader), 7th February, 1958; T. J. Orr (024805), 26th November, 1958.

Active Citizen Air Force: Medical Branch.

The heading in the notification regarding the transfer from the Reserve of Pilot Officers R. J. Chapman (026761) and A. J. Reading (026836) as approved in Executive Council Minute No. 65 of 1955, appearing in *Gazette* No. 46, dated 22nd September, 1955, is amended to read Sydney University Squadron.

Air Force Reserve: Medical Branch.

The following are appointed to a commission with the rank of Flight Lieutenant: Zygfryd Atlas (257937), 1st March, 1955; Peter John Bayliss (036611), 1st August, 1955.

The following Air Cadets are appointed to a commission, provisionally, with the rank of Pilot Officer: Robert Graeme Cameron (036604), Robert Hugo Mackay (036622), 1st August, 1955; John Thomas Goodchild Renney (04791), Lloyd Stewart Coats (04783), Malcolm David Silver (041265), John Edward Stacey Alwyn (04778), Edwin Walter Knight (052512), Keith Walter Grote (04787), Robert John Klaebe (04795), 17th September, 1955.

Flying Officer D. S. Forbes (439694) is transferred from the General Duties Branch and is promoted to the rank of Flight Lieutenant, 7th September, 1955.

Notice.

RADIATION BIOLOGY CONFERENCE.

UNDER the auspices of the Cancer Institute Board and the Anti-Cancer Council of Victoria a conference in radiation biology will be held at the Cancer Institute, 483 Little Lonsdale Street, Melbourne, from December 12 to 16, 1955. Professor W. V. Mayneord, of the Institute of Cancer Research, Royal Cancer Hospital, London, and Dr. F. G. Spear, of the Strangeways Research Laboratory, Cambridge, will deliver lectures at the conference.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Lilley, Leonard Bruce, M.B., B.S., 1954 (Univ. Sydney), Royal Newcastle Hospital, Newcastle.

Briscoe, Mary, M.B., B.S., 1954 (Univ. Sydney), District Hospital, Kurri Kurri, New South Wales.

Congresses.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

THE sixth International Congress of Otolaryngology will be held in Washington, D.C., from Sunday, May 5, to Friday, May 10, 1957. Selected subjects for the plenary sessions will be chronic suppuration of the temporal bone, collagen disorders of the respiratory tract and papilloma of the larynx. Communications of two types are invited: contributions to the discussion of the selected subjects (speakers limited to five minutes); original papers (speakers limited to fifteen minutes). All communications should be in one of the official languages—English, French, German, Spanish. Motion pictures will be shown continuously except during plenary sessions. There will be both scientific and technical exhibits. Those wishing to submit contributions to the programme should communicate with the General Secretary.

The subscription for members is $25.00 (U.S.A.). This includes the privilege of attendance at all official Congress meetings except the banquet for which an additional charge will be made. Other persons accompanying members may be registered as associates at a fee of $10.00 (U.S.A.).

All communications should be addressed to the General Secretary, Paul H. Holinger, M.D., 700 No. Michigan Avenue, Chicago II, Illinois, United States of America.

Deaths.

THE following death has been announced:

LEY.—Mark Aloysius Ley, on November 17, 1955, at Melbourne.

Diary for the Month.

DEC. 12.—Victorian Branch, B.M.A.: Executive of Branch Council.
DEC. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DEC. 14.—Victorian Branch, B.M.A.: Branch Council.
DEC. 20.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

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